

Podrid's Real-World

ECGs

A Master's Approach
to the Art and Practice
of Clinical ECG Interpretation

Volume 5 **Narrow and Wide Complex
Tachyarrhythmias and Aberration** **Part B: Practice Cases**

Philip Podrid, MD • Rajeev Malhotra, MD, MS

Rahul Kakkar, MD • Peter A. Noseworthy, MD

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Volume 5 **Narrow and Wide Complex Tachyarrhythmias and Aberration—Part B: Practice Cases**

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Practice Cases

Practice Case 62

A man is brought to the emergency department having been found unconscious. Prior to his arrival, the emergency medical technicians faxed a 12-lead ECG to you (ECG 62A). The history they relay on route is that of a 68-year-old man with a history of a remote myocardial infarction who was found by his family unconscious on the floor of his apartment. He had been well the morning of presentation.

ECG 62A



Practice Case 62

He was treated in the field based on the ECG provided. Initial vital signs were notable for a tachycardia and hypotension, and after appropriate therapy, his vital signs stabilized. He was intubated prior to arrival.

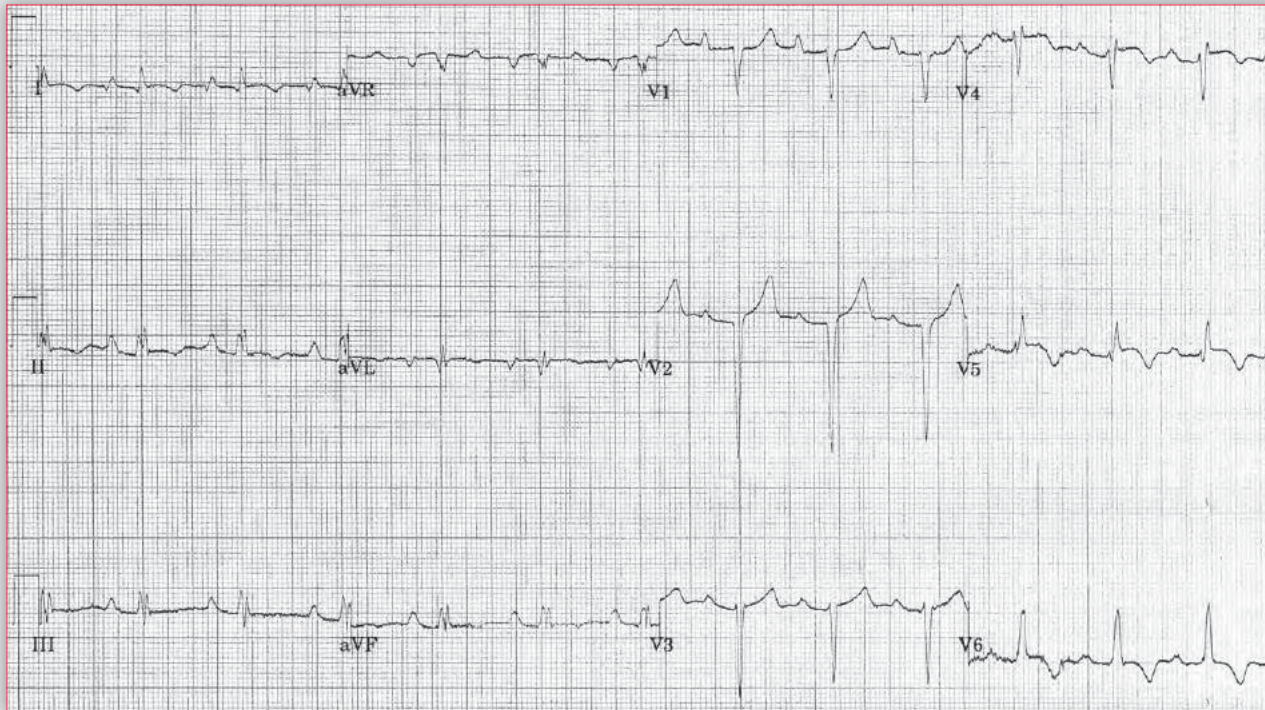
Upon admission to the emergency department, a repeat ECG is obtained (ECG 62B).

What is your interpretation of his initial ECG?

What arrhythmia diagnosis is suggested?

What clinical diagnosis is suggested in comparing the two tracings?

ECG 62B



Podrid's Real-World ECGs



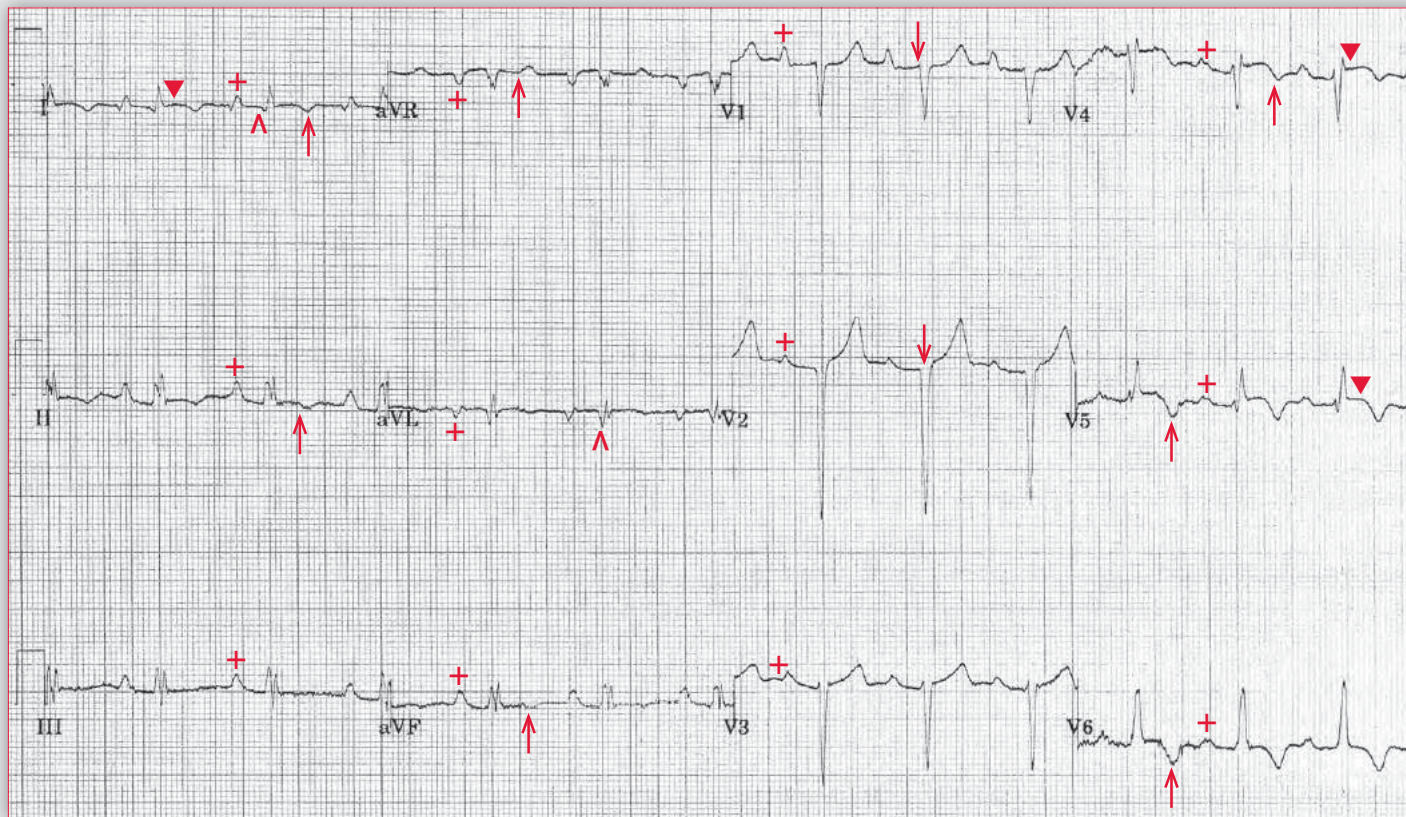
ECG 62A Analysis: Ventricular tachycardia (sustained monomorphic)

ECG 62A shows a regular rhythm at a rate of 220 bpm. The QRS complex duration is increased (0.18 sec), and the morphology is not typical for either a right or left bundle branch block. Although there are no obvious P waves seen, there are inconsistent abnormalities of the ST-T waves, best seen in leads I (↑) and V1 (↓). The variability in the ST-T waves may be due to superimposed P waves or may be differences in repolarization. There is also variability in the QRS complex morphology seen in lead V1 (Λ). Such variability is not seen in a supra-ventricular tachycardia in which depolarization and repolarization are

uniform, as the activation of the ventricle follows the same pathway each time. Thus, every QRS complex and ST-T wave is the same. In ventricular tachycardia, the activation of the left ventricle is not using the normal His-Purkinje system, but rather an alternative pathway that results in direct ventricular activation. The activation sequence may be variable, accounting for differences in the QRS complex. The abnormalities in ventricular depolarization is associated with abnormalities in ventricular repolarization, accounting for the ST-T wave changes.

continues

Podrid's Real-World ECGs



ECG 62B Analysis: Normal sinus rhythm, right atrial hypertrophy (abnormality), first-degree AV block (prolonged AV conduction), old lateral and anteroapical myocardial infarction, left ventricular aneurysm, low voltage in limb leads

ECG 62B is from the same patient as ECG 62A. It is the baseline ECG for the patient. There is a regular rhythm at a rate of 78 bpm. There is a P wave before each QRS complex (+) and the PR interval is constant (0.26 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm with a first-degree AV block (prolonged AV nodal conduction). The P waves are tall and peaked in leads II and aVF and all positive in lead V1, consistent with right atrial hypertrophy or a right atrial abnormality. The QRS complex duration is normal (0.08 sec) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). There is low QRS voltage in the limb leads (< 5 mm in each lead). The

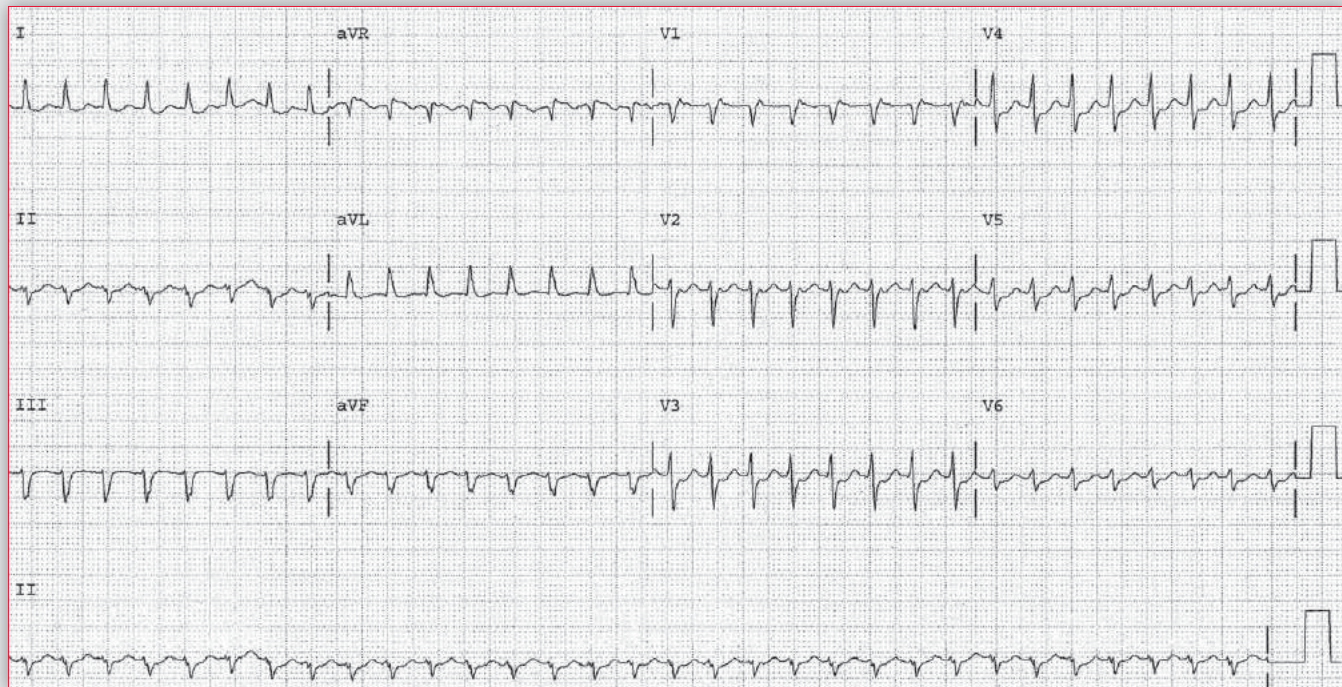
QT/QTc durations are normal (380/430 msec). There are Q waves in leads I and aVL (^), consistent with a lateral wall myocardial infarction. There is also a QS complex in leads V1–V2 (↓), suggestive of an anteroseptal myocardial infarction. Seen in leads I and V4–V5 (▼) are ST-segment elevations, suggesting that there is a left ventricular aneurysm as a result of the previous infarction. Lastly, there are T-wave abnormalities (†) in leads I, II, aVR, aVF, and V4–V6.

Sustained monomorphic ventricular tachycardia in a patient with a previous myocardial infarction and a left ventricular aneurysm is related to scar with resultant reentrant circuit and is not a result of ischemia. ■

Practice Case 63

A 49-year-old woman presents for a first visit to her primary care physician. She has no significant medical history, but does state that several months before the visit she was seen in the emergency department because of the acute onset of palpitations with a tachycardia. She states that the tachycardia

ECG 63A



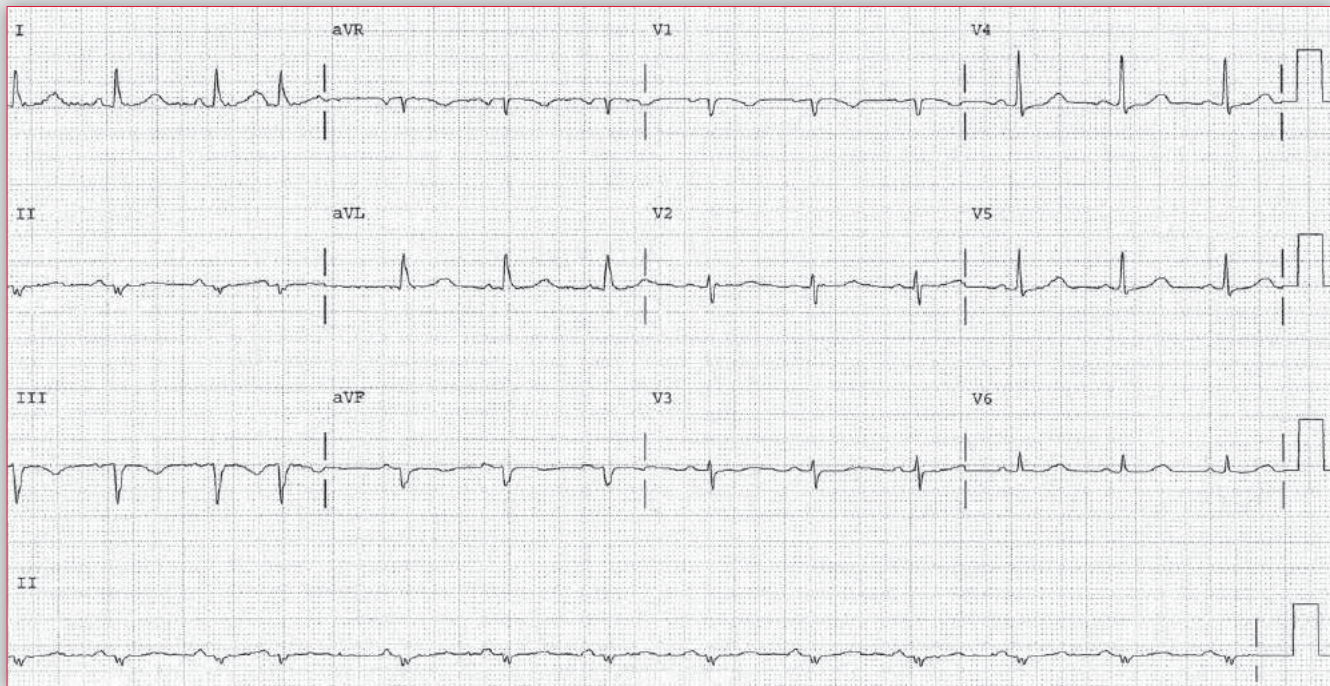
Practice Case 63

abruptly stopped after she received an intravenous medication, but she does not know what was given. She does bring an ECG that was obtained at the time (ECG 63A). Her physical examination is normal. A new ECG is obtained (ECG 63B) and is compared to the ECG from the emergency department.

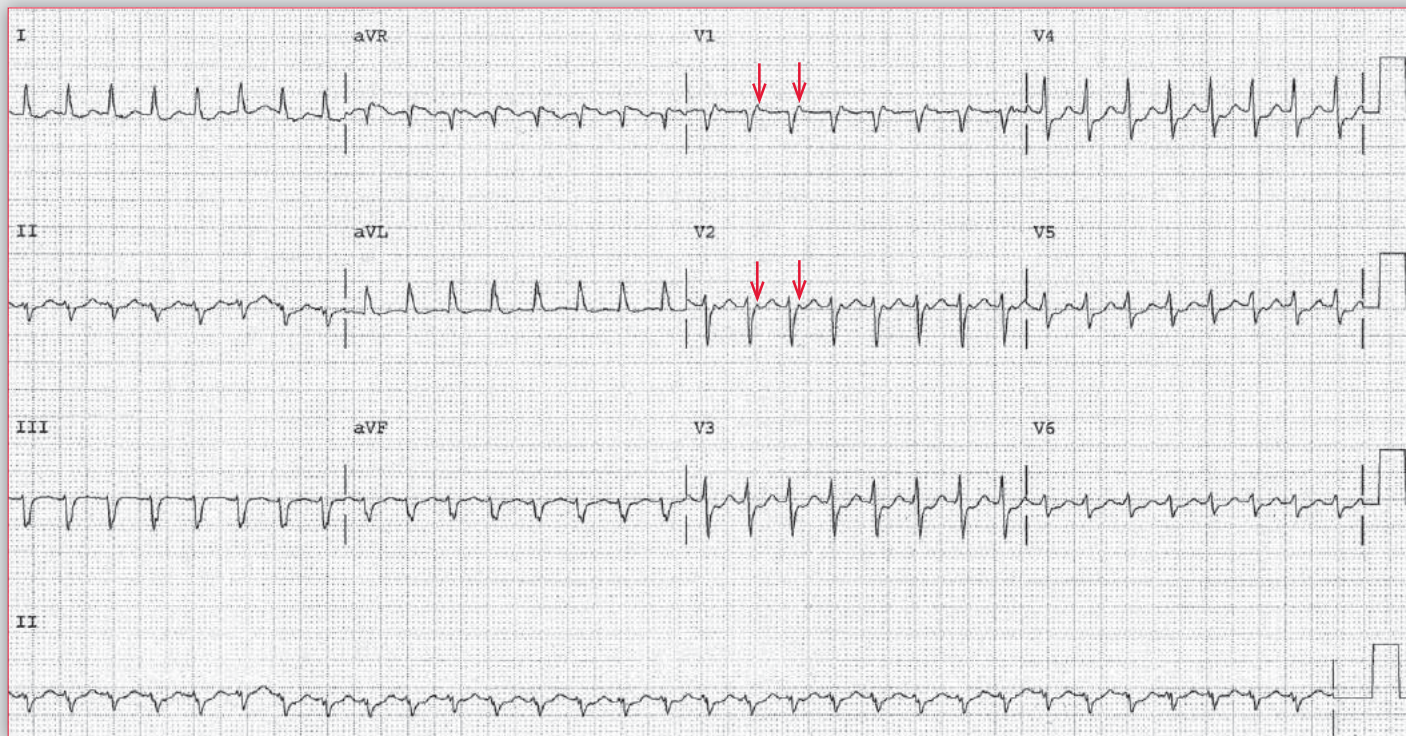
What do the ECGs show?

What is her diagnosis?

ECG 63B



Podrid's Real-World ECGs



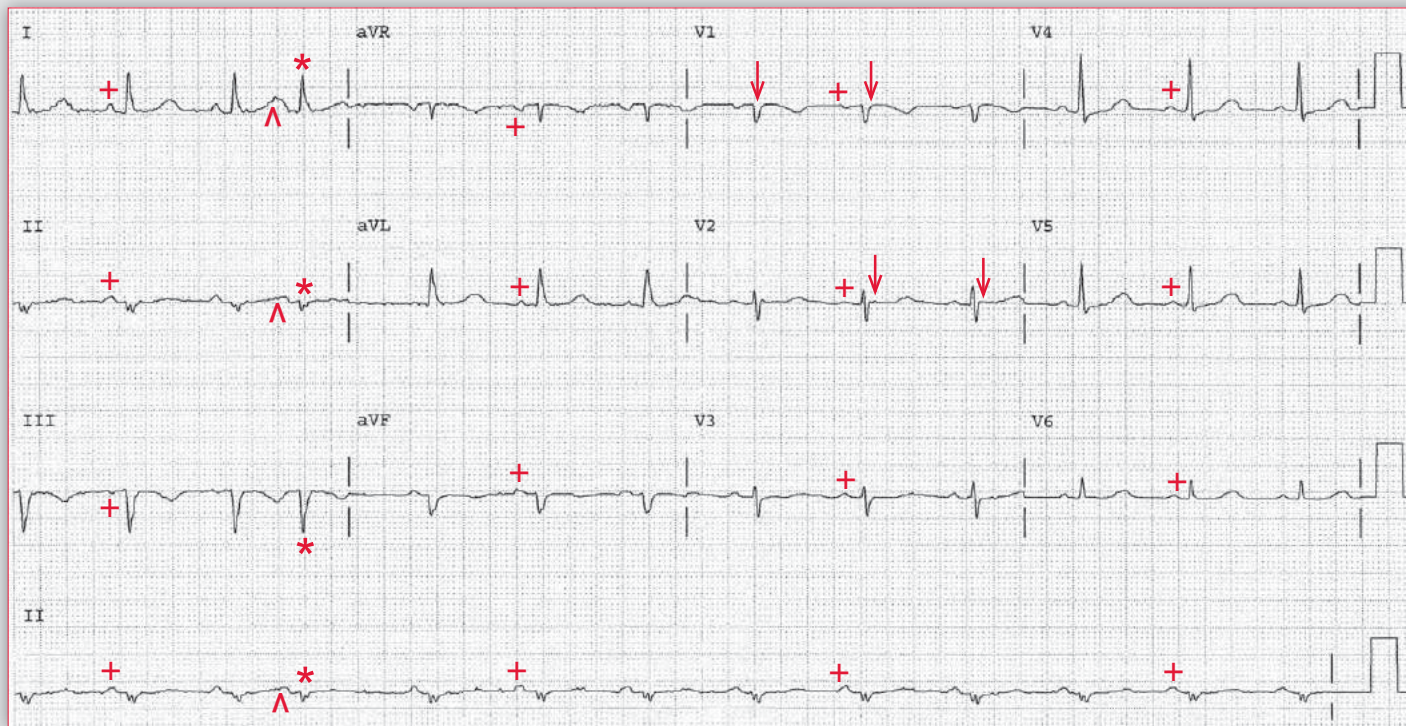
ECG 63A Analysis: Narrow complex supraventricular tachycardia (no-RP tachycardia), atrioventricular nodal reentrant tachycardia, left anterior fascicular block, low voltage

ECG 63A shows a regular rhythm at a rate of about 190 bpm. The QRS complex duration is normal (0.08 sec), and there is low QRS voltage throughout (< 5 mm in each limb lead and < 10 mm in each precordial lead). The axis is extremely leftward between -30° and -90° (positive QRS complex in leads I and negative in leads II and aVF). There are two etiologies for an extreme left axis, *ie*, an old inferior wall myocardial infarction in which there is a deep Q wave in leads II and aVF, or a left anterior fascicular block in which there is an rS morphology in leads II and aVF. Hence this is a left anterior fascicular block. The QT/QTc intervals are normal (240/430 msec).

There are no obvious P waves seen before or after any of the QRS complexes. However, there is a small R' seen in leads V1–V2 (\downarrow), at the terminal part of the QRS complex. This may be the normal QRS morphology or might be a superimposed P wave. This can only be established by comparison of the QRS complex morphology during normal sinus rhythm. Therefore, this might be termed a no-RP tachycardia, and the most common etiology is a typical or common atrioventricular nodal reentrant tachycardia (slow-fast) in which there is simultaneous (or almost simultaneous) antegrade ventricular activation via the slow pathway and retrograde atrial activation via the fast pathway.

continues

Podrid's Real-World ECGs



ECG 63B Analysis: Normal sinus rhythm, left anterior fascicular block

ECG 63B is from the same patient as ECG 63A. There is a regular rhythm at a rate of 72 bpm. There is a P wave (+) before all of the QRS complexes with a stable PR interval (0.16 sec). The QRS complex duration, morphology, and axis are identical to that seen in ECG 63A. The QT/QTc intervals are normal (390/430 msec). There is one premature complex (*) that is preceded by a premature P wave (^); the QRS complex morphology is identical to all of the other QRS complexes. Hence this is a premature atrial complex.

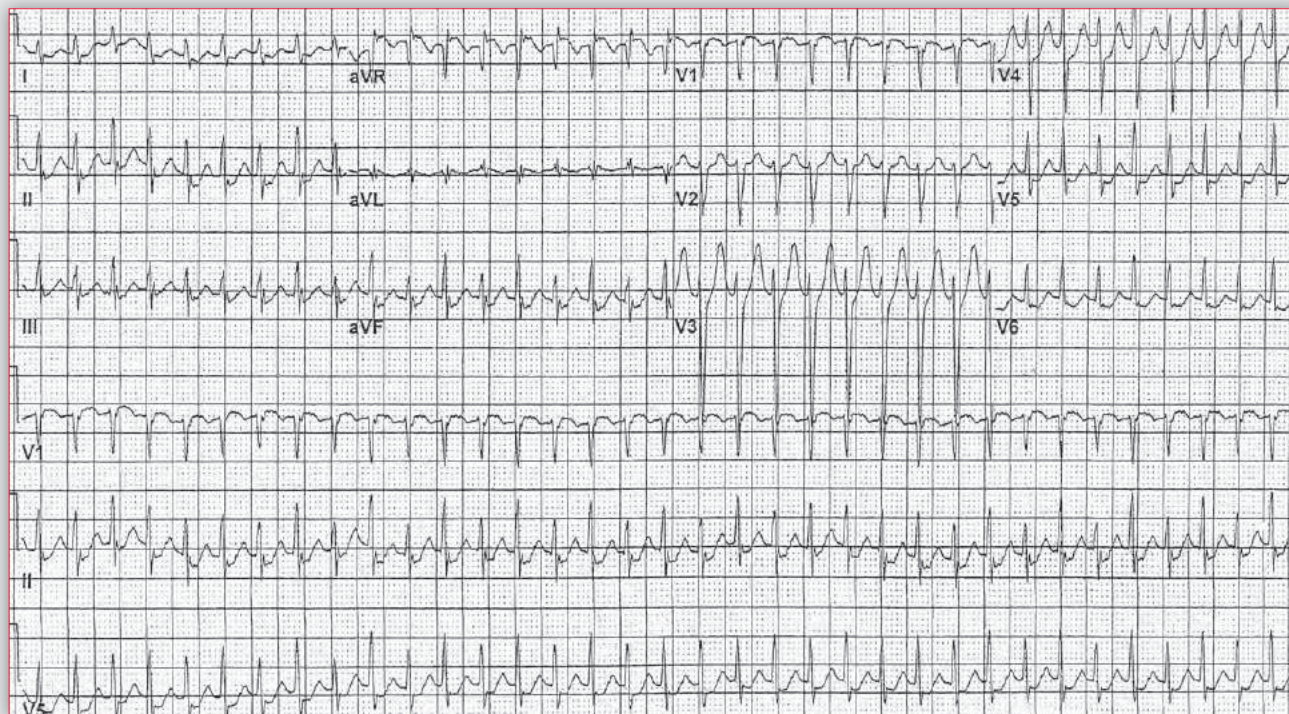
Noted is the absence of the R' in leads V1–V2 (↓), suggesting that this was indeed the P wave superimposed on the terminal portion of the QRS complex. This further supports the diagnosis of a typical or common atrioventricular nodal reentrant tachycardia. ■

Practice Case 64

A 25-year-old woman presents to your office because of episodes of palpitations that she has been able to break by coughing. Although she does not have any known heart disease, the palpitations have been occurring every few months for the past several years.

She does remember that when the palpitations first occurred, an ECG was obtained and she was told of some abnormality, but does not remember what it was. Physical examination is normal and an ECG is interpreted as normal. A Holter monitor is ordered.

ECG 64A



Practice Case 64

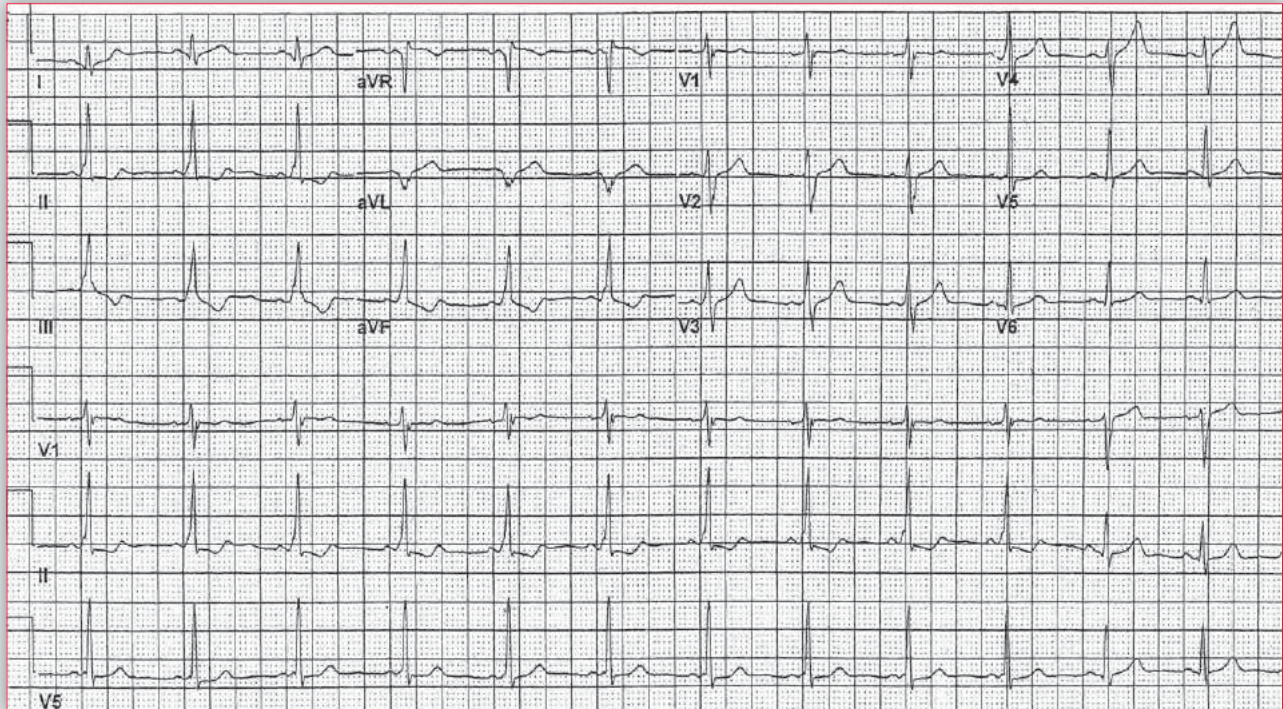
Prior to having this study, she again has an episode of palpitations and presents to the emergency department. An ECG is obtained (ECG 64A). She is treated for the tachycardia and a follow-up ECG is recorded (ECG 64B).

What is the etiology of the tachycardia shown in ECG 64A?

What is the underlying diagnosis and how does ECG 67B help prove this?

What therapies are available for long-term treatment?

ECG 64B



Podrid's Real-World ECGs



ECG 64A Analysis: Short RP tachycardia, orthodromic AVRT, electrical alternans

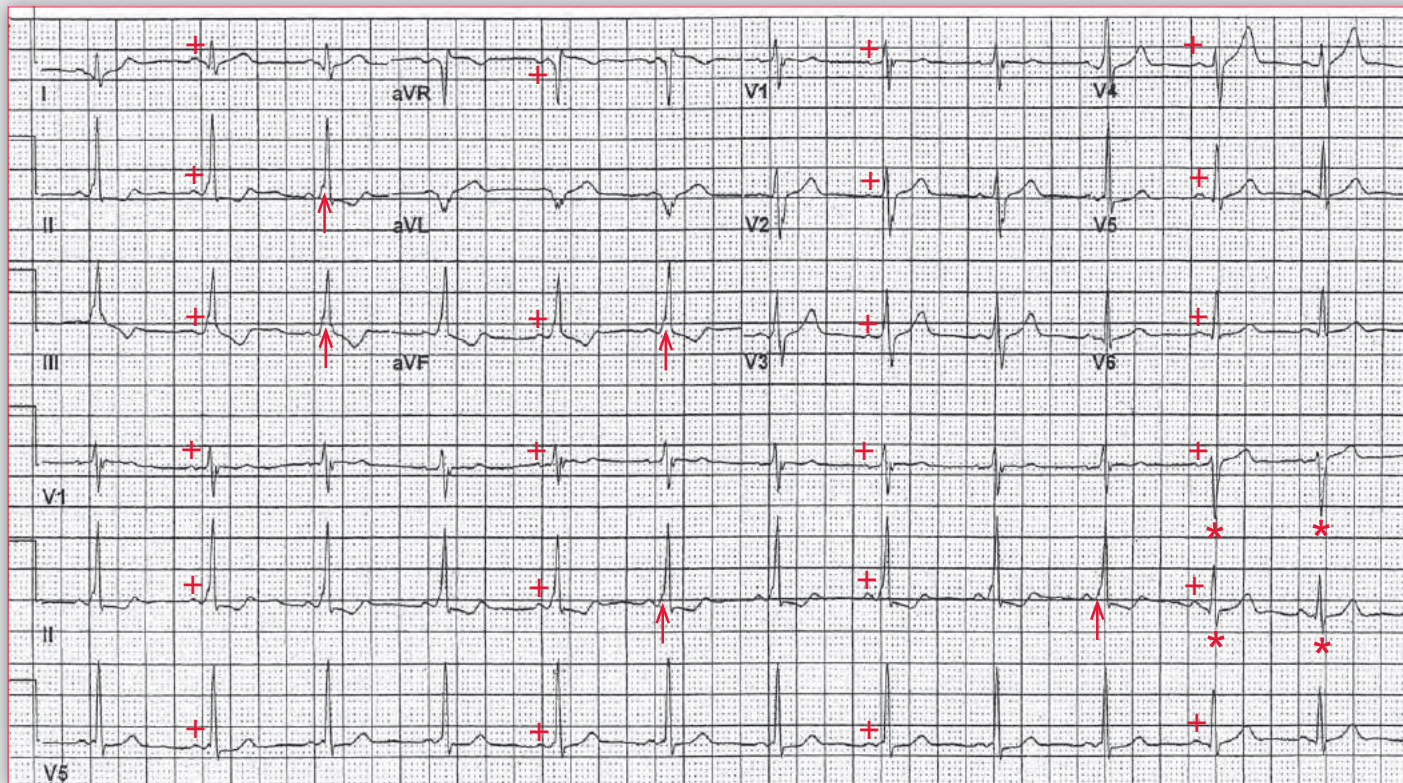
ECG 64A shows a regular rhythm at a rate of about 220 bpm. The QRS complex duration is normal (0.08 sec). There is a normal morphology and axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (220/420 msec). Noted are alternating changes in the QRS complex amplitude (\wedge ,+), especially prominent in leads II, aVR, aVF, and V4–V5. This is termed QRS or electrical alternans. This can be seen with any rapid supraventricular tachycardia and is the result of beat-to-beat changes in calcium fluxes into the ventricular myocardium.

There are no clear P waves seen before or after any of the QRS complexes. However, there is notching of the ST segments, especially prominent in leads I, II, V1, and V6 (\uparrow). When comparing these QRS

complexes to those in the baseline ECG (ECG 64B), it can be seen that these subtle notches within the ST segment are not present at baseline and therefore are likely to be P waves. This is, therefore, a short RP tachycardia (RP interval = 0.12 sec and PR interval = 0.20 sec). Etiologies include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atrioventricular reentrant tachycardia, and typical or common atrioventricular nodal reentrant tachycardia of an unusual type (slow-slow). The ECG does not provide any further information to establish the etiology. However, this would be an usual presentation for sinus tachycardia or atrial flutter, which at this rate would be with 1:1 AV conduction.

continues

Podrid's Real-World ECGs



ECG 64B Analysis: Normal sinus rhythm, Wolff-Parkinson-White syndrome

ECG 64B is from the same patient as ECG 64A. There is a regular rhythm at a rate of 76 bpm. There are P waves before each QRS complex with a stable and short PR interval 0.12 sec. The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm.

The QRS complex duration is prolonged (0.12 sec) as a result of a slowed or “slurred” upstroke (↑); this is termed a delta wave. The QT/QTc intervals are normal (360/140 msec and 360/410 msec when the prolonged QRS complex duration is considered). The short PR interval and prolonged QRS complex duration due to a delta wave is typical for a preexcitation pattern, specifically a Wolff-Parkinson-White pattern. However, the delta wave is not very pronounced and is obvious primarily only in leads II, III, and aVF. The ECG changes are subtle and the presence of a WPW pattern could certainly be overlooked. Here, Q waves in leads I and aVL (^) as well as a positive delta wave in lead V1 are consistent with a left lateral bypass tract. The last two QRS complexes (*) have a normal PR interval (0.18 sec) and normal QRS complex duration without a delta wave. Hence these are normal QRS complexes without evidence for preexcitation. These QRS complexes have a duration and morphology that is identical to those in ECG 64A. The fact that there is spontaneous normalization of the

QRS complex, indicating that there is no impulse conduction via the accessory pathway, means that the WPW pattern is intermittent, which accounts for the initial ECG that was normal and without evidence of preexcitation.

The presence of a WPW pattern during sinus rhythm confirms the fact that the short RP tachycardia in ECG 64B is an atrioventricular reentrant tachycardia. As the QRS complex during the tachycardia is narrow and normal in morphology, this is an orthodromic atrioventricular reentrant tachycardia. In this situation, the antegrade conduction to activate the ventricles is via the normal AV node–His-Purkinje system, and retrograde conduction to activate the atria is via the accessory pathway. Thus, the last two QRS complexes (*) are the result of normal conduction via the AV node–His-Purkinje system and are identical in morphology to the QRS complex during the orthodromic AVRT as they are also conducted through the AV node–His-Purkinje pathway.

Therapy for WPW can be pharmacologic, with an AV nodal blocking agent, or non-pharmacologic, *ie*, radiofrequency ablation. As this young patient has symptomatic arrhythmia, most often therapy would be accessory pathway ablation. ■

Practice Case 65

An 80-year-old woman with a severe dilated cardiomyopathy presents because of worsening shortness of breath. Her blood pressure is normal, but she is noted to be tachycardic. An ECG is obtained (ECG 65A). It is compared to his baseline ECG (ECG 65B).

ECG 65A

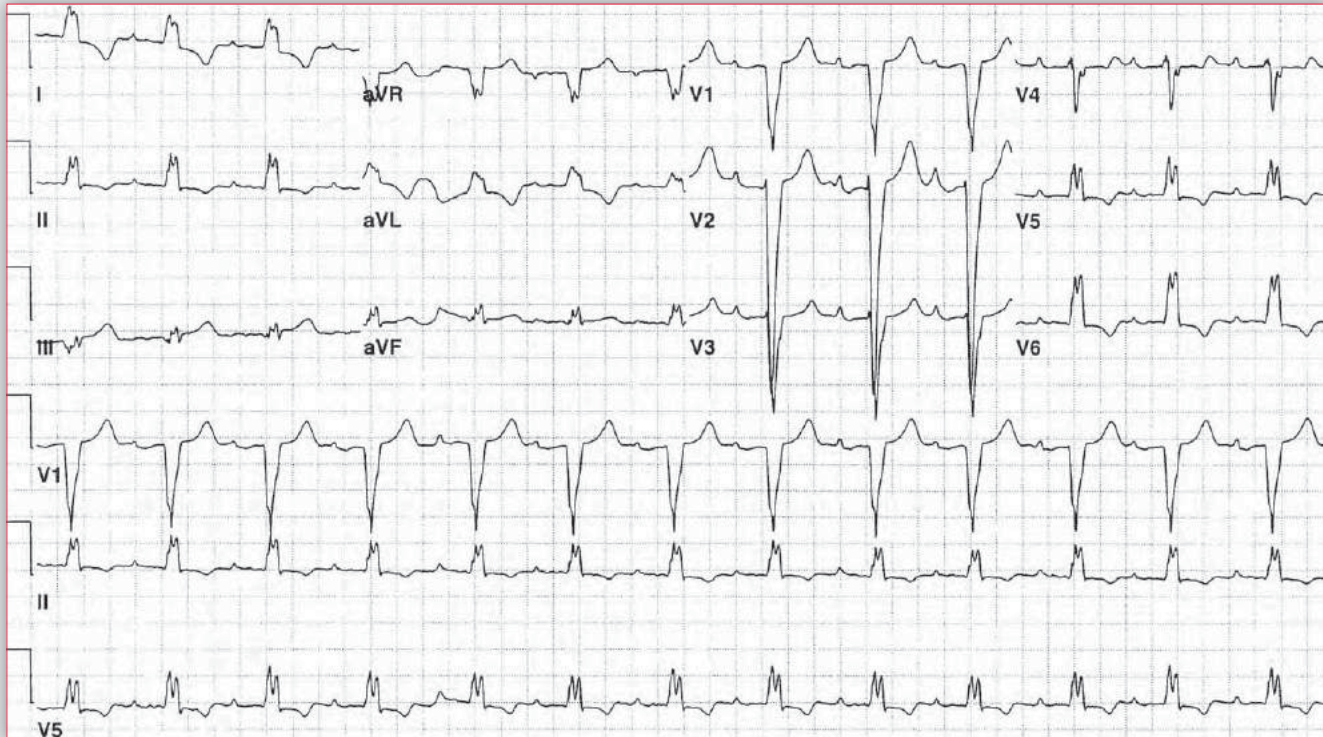


Practice Case 65

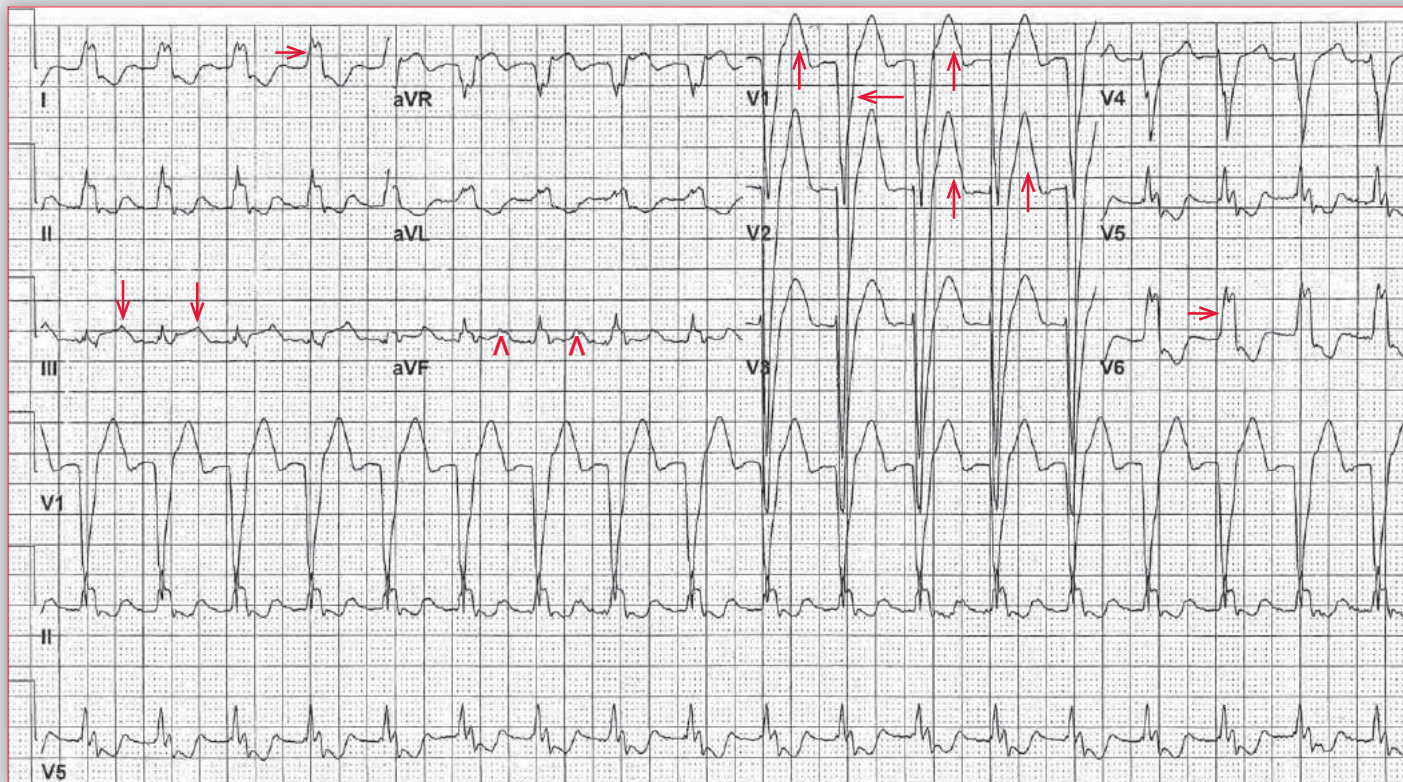
What do these ECGs show?

What is the diagnosis?

ECG 65B



Podrid's Real-World ECGs



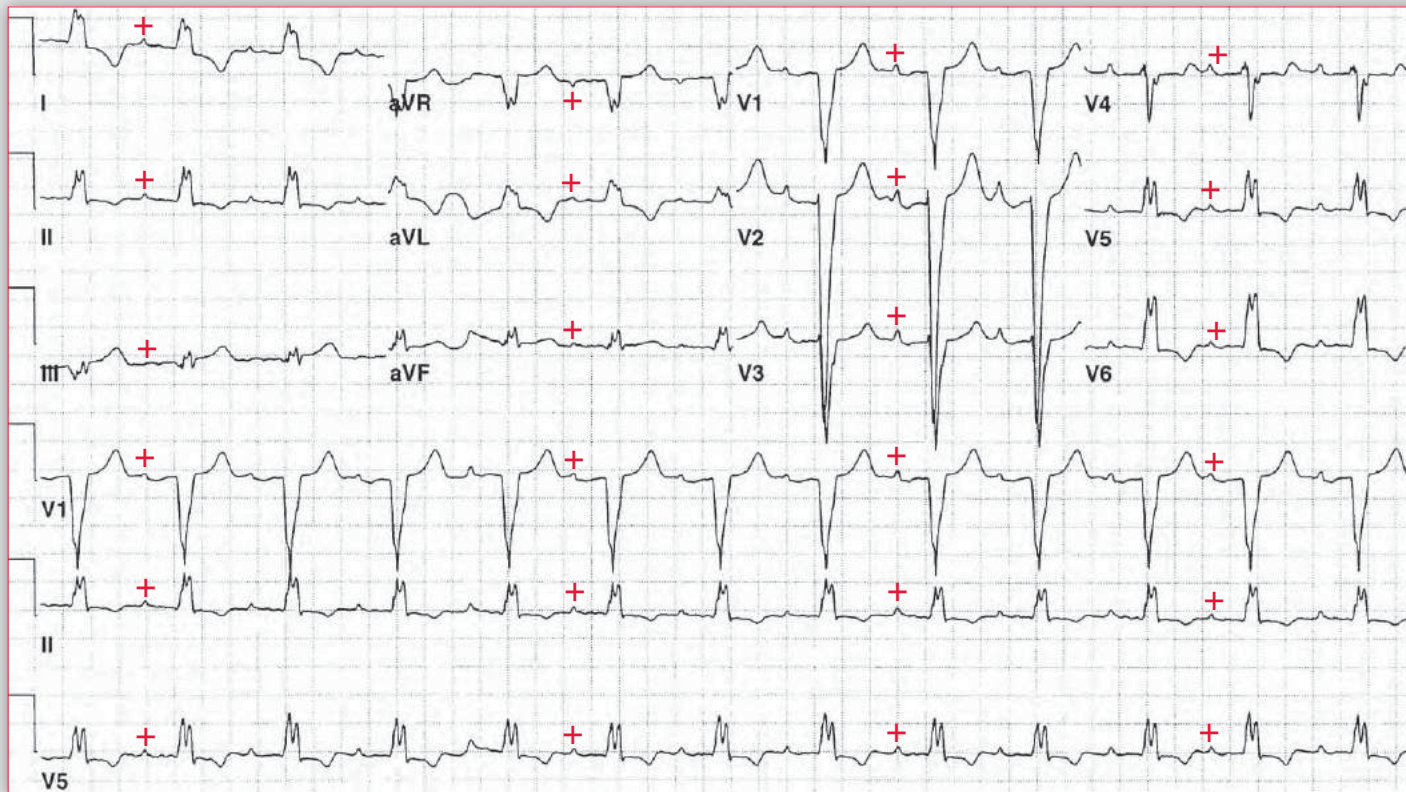
ECG 65A Analysis: Wide complex tachycardia, long PR tachycardia, sinus tachycardia, first-degree AV block (prolonged AV conduction), left bundle branch block

ECG 65A shows a regular rhythm at a rate of 112 bpm. The QRS complex duration is increased (0.16 sec), and the morphology is typical for a left bundle branch block with broad R wave in leads I and V6 (→) and a deep QS complex in lead V1 (←). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (380/520 msec) but are normal when corrected for the prolonged QRS complex duration (320/430 msec). There are ST-T wave changes secondary to the left bundle branch block.

Although there are no obvious P waves, it can be seen that there is a notching of the downslope of the T wave, well seen in leads V1 and V2 (↑). Also noted is a sharp peak of the T wave in lead III (↓) and an abnormality of the T wave in lead aVF (^). T waves should have a smooth upstroke and downstroke. Any notches, ridges, bumps or other irregularity on the T wave suggests a superimposed P wave. The P wave appears to be positive in these leads. The PR interval is stable (0.24 sec). This is a supraventricular tachycardia, although the etiology is not certain. It is possibly a sinus tachycardia or an atrial tachycardia.

continues

Podrid's Real-World ECGs



ECG 65B Analysis: Normal sinus rhythm, first-degree AV block, left bundle branch block

ECG 65B is from the same patient as ECG 65A. There is a regular rhythm at a rate of 76 bpm. The QRS complex duration, morphology, and axis are identical to the QRS complexes in ECG 65A, and there is a left bundle branch block morphology with a normal axis. The QT/QTc intervals are the same as ECG 65A and are normal (440/500 msec and 380/430 msec when the prolonged QRS complex duration is considered). There is a P wave before each QRS complex (+) with a stable PR interval (0.24 sec), which is identical to the PR interval seen in ECG 65A. The P wave is positive in leads I, II, aVF, and

V4–V6. Hence this is a normal sinus rhythm and confirms that the rhythm in ECG 65A is a sinus tachycardia. There is an underlying left bundle branch block present during normal sinus rhythm that persists during sinus tachycardia. Sinus tachycardia is very commonly seen with decompensated heart failure and may be one of the more sensitive markers of early heart failure, as this condition is associated with activation of the sympathetic nervous system. With therapy for heart failure, the heart rate slows, and this indicates improvement in hemodynamic status. ■

Notes

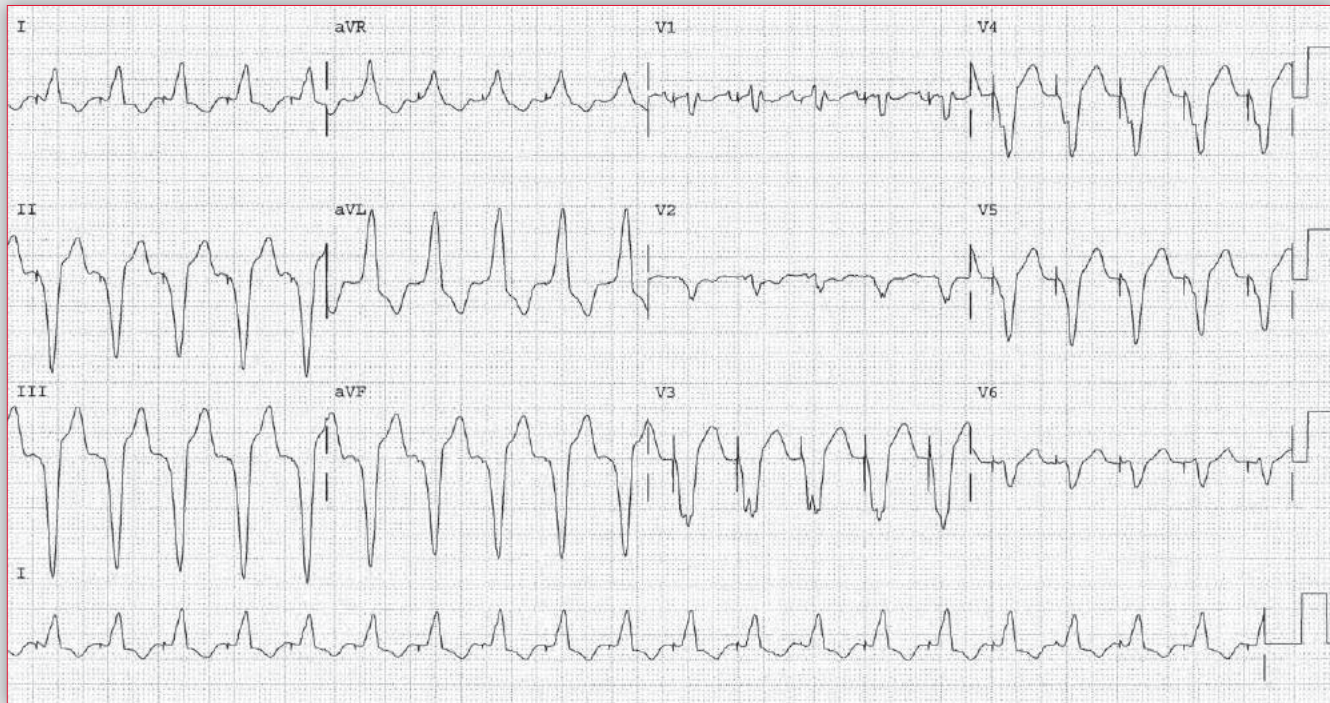
Practice Case 66

A 68-year-old man who had a pacemaker placed for complete heart block presents to his physician with a racing heart.

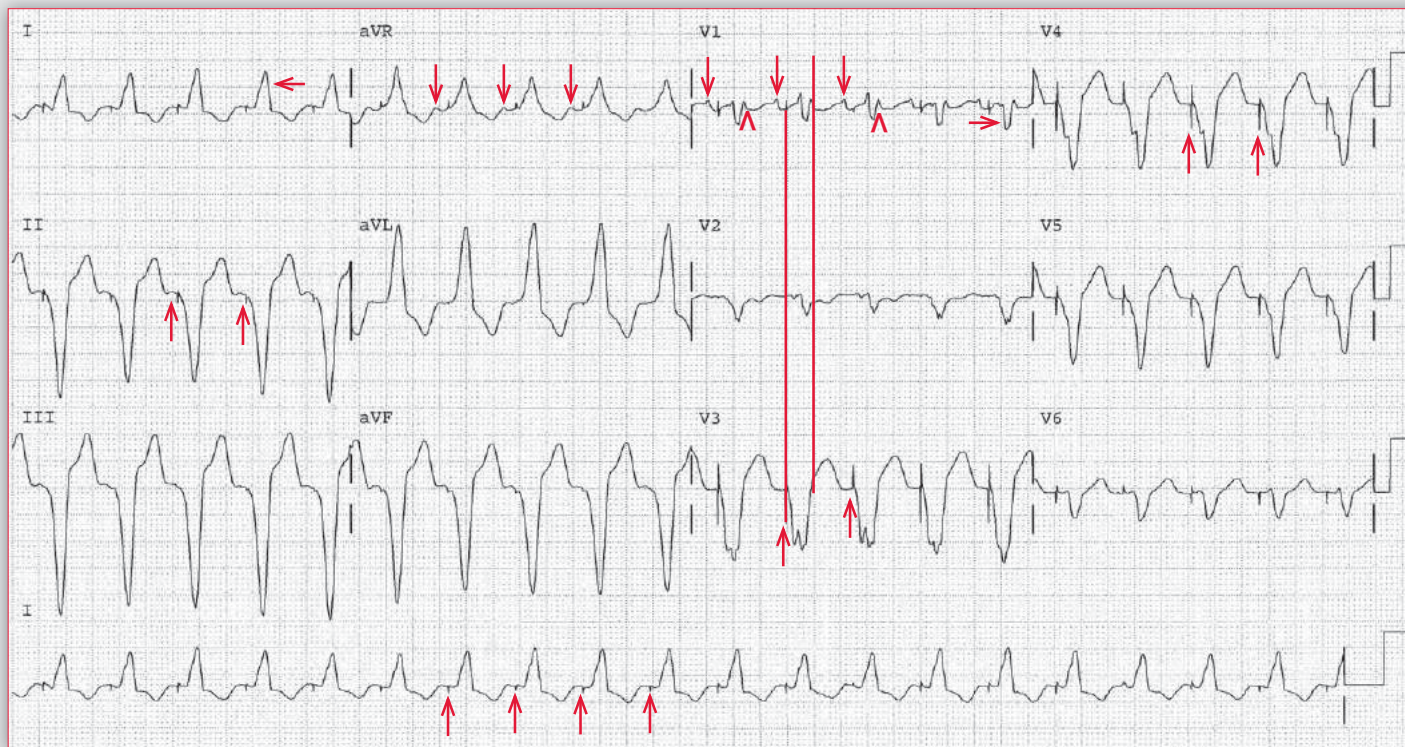
What does the ECG show?

What is the underlying mechanism of the patient's symptoms?

How can this be treated?



Podrid's Real-World ECGs



ECG 66 Analysis: Pacing-associated tachycardia, atrial flutter tracking with 2:1 conduction

ECG 66 shows a regular rhythm at a rate of 128 bpm. The QRS complex duration is increased (0.18 sec). A pacemaker stimulus can be seen before each QRS complex (†). It should be noted that the paced QRS complexes have a left bundle branch block morphology, especially a broad R wave in lead I (←) and also a QS morphology in lead V1 (→). The QRS complexes also have a QS morphology across the precordium. Hence the ventricular pacing electrode is in the right ventricle. As the rate is 128 bpm, this is a pacemaker-associated wide complex tachycardia, *ie*, the pacemaker is tracking a rapid atrial rate. Single-chamber pacemakers, which are most commonly programmed for a demand mode, are activated whenever the intrinsic heart rate falls below the lower rate limit of the pacemaker. The pacemaker will pace at its programmed rate, *ie*, the lower rate limit of the pacemaker. In this case, the pacemaker is pacing at a rate of 128 bpm, meaning that the pacemaker is responding to sensed atrial activity. Thus, this is a dual-chamber pacemaker with a lead in the right atrium and right ventricle. Close examination of lead V1 (as well as leads II and aVR) demonstrates an atrial waveform before each pacemaker stimulus (↓). Hence the pacemaker is functioning in a mode of P wave (atrial) sensing or P-wave synchronous ventricular pacing. However, it can be seen that there is a second atrial waveform (^) at the very end of the QRS complex in lead V1 that is on time. Although it might be felt that this positive waveform at the end of the QRS complex is actually part of this complex; when the QRS complex width in another lead is measured from the pacing spike to the end of the QRS complex (||), it can be seen that this positive waveform is superimposed at the terminal

portion of the complex. In V1, the initial portion of the QRS complex is isoelectric, and it can be seen that there appears to be a slight delay after the pacemaker stimulus to the onset of the QRS complex. Hence there is a regular atrial rate of almost 260 bpm, and the underlying atrial rhythm is atrial flutter. As this is a dual-chamber pacemaker, the atrial lead is tracking the atrial flutter. However, the pacemaker is unable to respond with a ventricular stimulus to every atrial impulse, as the atrial rate exceeds the upper rate limit of the pacemaker, *ie*, the fastest rate it is capable of sensing and hence of pacing. Thus, only every other atrial impulse is sensed, resulting in a ventricular stimulus and a rate of 128 bpm.

Acute treatment is to reduce the ventricular rate. However, AV nodal blocking agents will not be effective, as the pacemaker is an additional AV connection, and it is responsible for the rapid rate and not AV nodal conduction. Hence the initial therapy would be to disable all atrial sensing, so that the pacemaker is unable to sense and track the atrial arrhythmia and, therefore, all conduction will be via the AV node. This can be achieved with a magnet that disables all atrial as well as ventricular sensing, *ie*, a DOO mode. However, as the atrial and ventricular pacemaker will function in a fixed-rate mode (and not an inhibited mode), there will be atrial and ventricular stimuli seen at the lower rate limit of the pacemaker, *ie*, absence of all sensing. These stimuli may capture the atrium or ventricle if they occur at an appropriate time such that the myocardium is responsive. ■

Notes

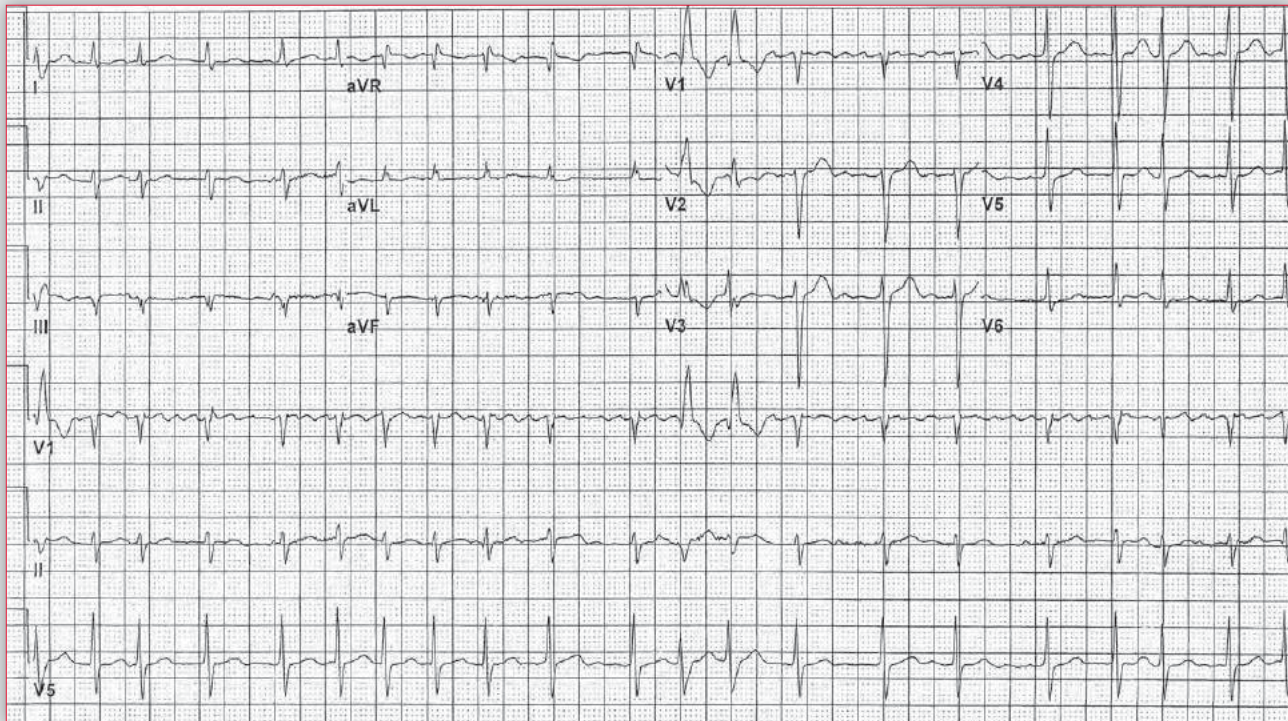
Practice Case 67

A 55-year-old man with atrial fibrillation is seen for a routine visit. An ECG is obtained, and his physician becomes concerned, as there are abnormal complexes seen. The patient is sent to a cardiologist for a consultation on the basis of this ECG.

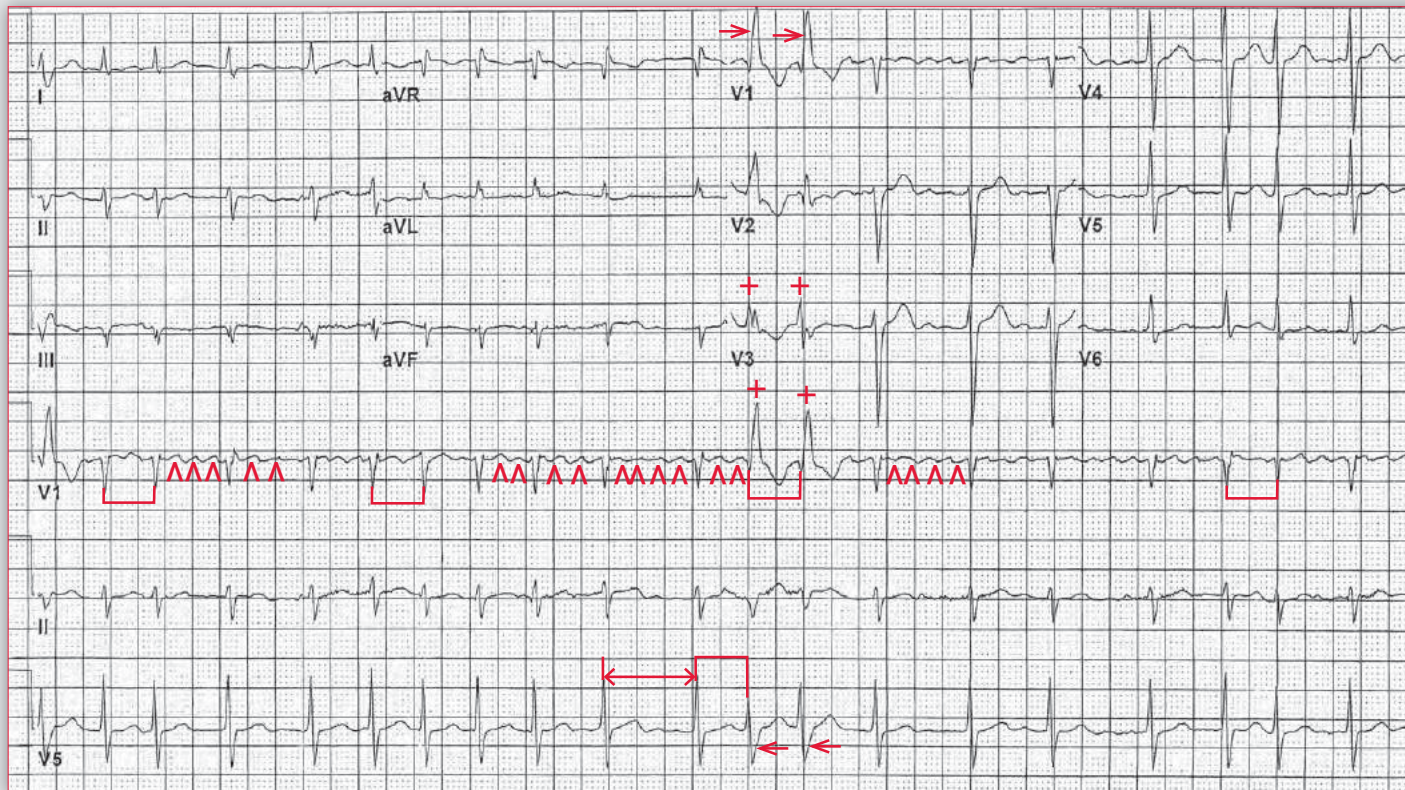
What does the ECG show?

What is the extent of his conduction disease?

What is the mechanism of the wide complex beats observed?



Podrid's Real-World ECGs



ECG 67 Analysis: Atrial fibrillation, left anterior fascicular block, low voltage in the limb leads, right bundle branch block aberration due to Ashman phenomenon

ECG 67 shows irregularly irregular rhythm as there is no pattern to the RR intervals. The average rate is 126 bpm. There are only three supraventricular rhythms that are irregularly irregular. These are (1) sinus arrhythmia in which there is one P-wave morphology and a stable PR interval, (2) multifocal atrial rhythm (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm) in which there are > 3 different P-wave morphologies and PR intervals and no clear dominant P-wave morphology, and (3) atrial fibrillation in which there are no organized P waves. In this ECG, there are no obvious P waves seen, but there are rapid and irregular undulations of the baseline (^) that are particularly evidence in lead V1. These are fibrillatory waves and hence the rhythm is atrial fibrillation. The QRS complex duration is normal (0.08 sec) and the morphology is normal. The axis is extremely leftward between -30° and -90° (QRS complex positive in lead I and negative in leads II and aVF with an rS morphology). This is termed a left anterior fascicular block. The QT/QTc intervals are normal (300/430 msec). There is low QRS complex voltage in the limb leads (QRS complex < 5 mm in amplitude in each limb lead).

There are two QRS complexes that are wide (duration = 0.12 sec) and different morphology (+); these complexes are aberrated. They have an RSR' morphology in lead V1 (\rightarrow) and a broad terminal S wave in lead V5 (\leftarrow). This morphology is typical for a right bundle branch block. The right bundle branch block aberration is not rate-related, as

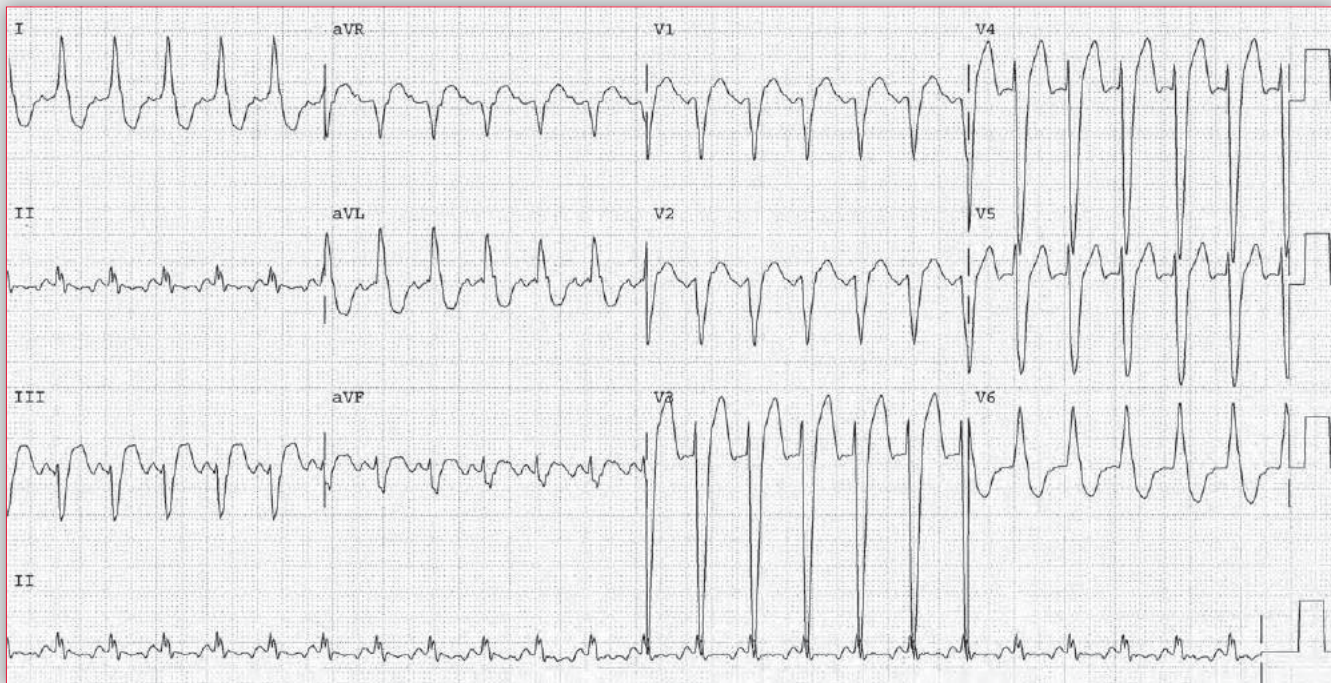
there are RR intervals that are equivalent to the RR intervals associated with the aberrated complexes (\square). However, note that preceding these aberrated complexes, there is a pattern of long-short RR intervals (\leftrightarrow , \square). Hence the aberration seen with these two QRS complexes is a result of an Ashman's phenomenon, which occurs as a result of normal physiologic rate-related changes of His-Purkinje refractoriness and not the result of conduction abnormalities of the His-Purkinje system. The refractory period of the His-Purkinje system prolongs at slower heart rates (longer RR intervals) and shortens at faster heart rates (shorter RR intervals). When there is an abrupt change in heart rate going from slow (long RR interval) to fast (short RR interval), His-Purkinje refractoriness does not adjust or adapt immediately. As refractoriness is still prolonged when the heart rate increases (short RR interval), one or several complexes are conducted with aberration. This is most commonly seen during atrial fibrillation as a result of the constantly changing RR intervals. Most commonly seen is a right bundle branch block aberration, probably because the baseline refractoriness of the right bundle is longer than that of the left bundle.

Not uncommonly, these complexes are confused with ventricular complexes, which appears to have been the concern in this case. As the occurrence of the Ashman phenomenon is a normal finding, there is no reason for any concern and no further evaluation or specific therapy is necessary. ■

Practice Case 68

A 49-year-old man with a prior myocardial infarction is admitted with dyspnea and a racing heart. He is found to be in congestive heart failure.

ECG 68A

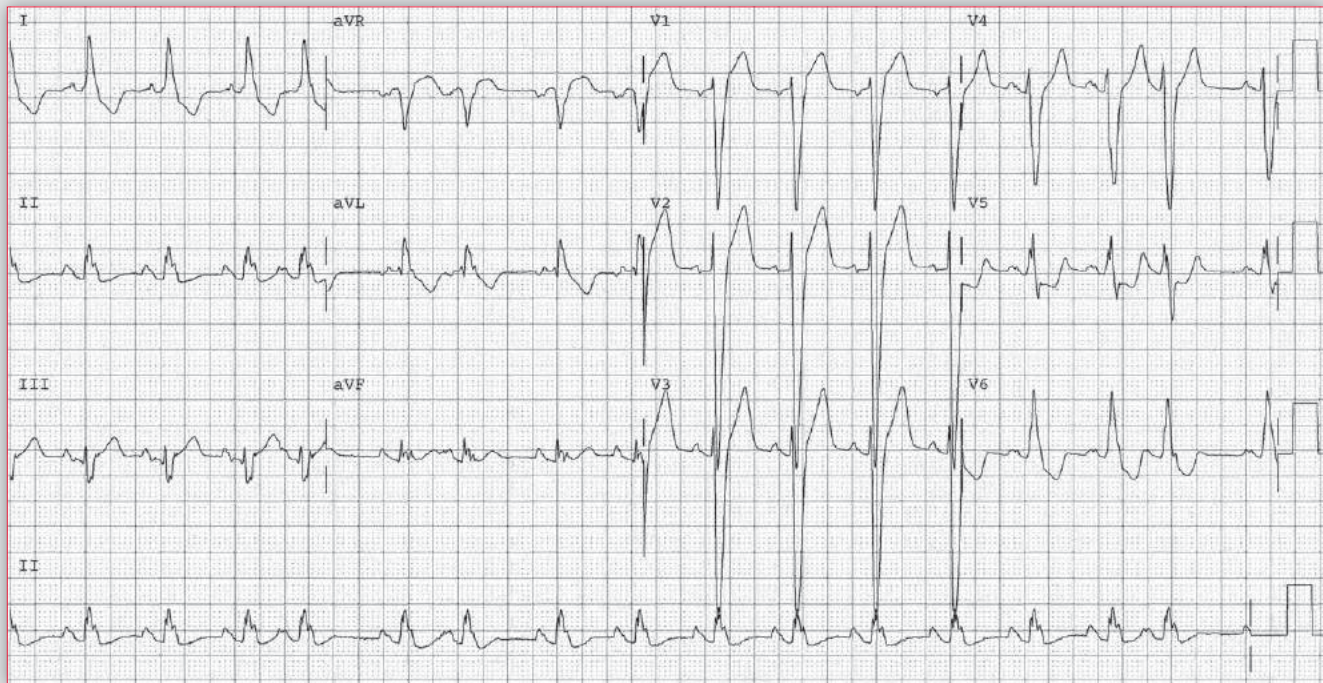


Practice Case 68

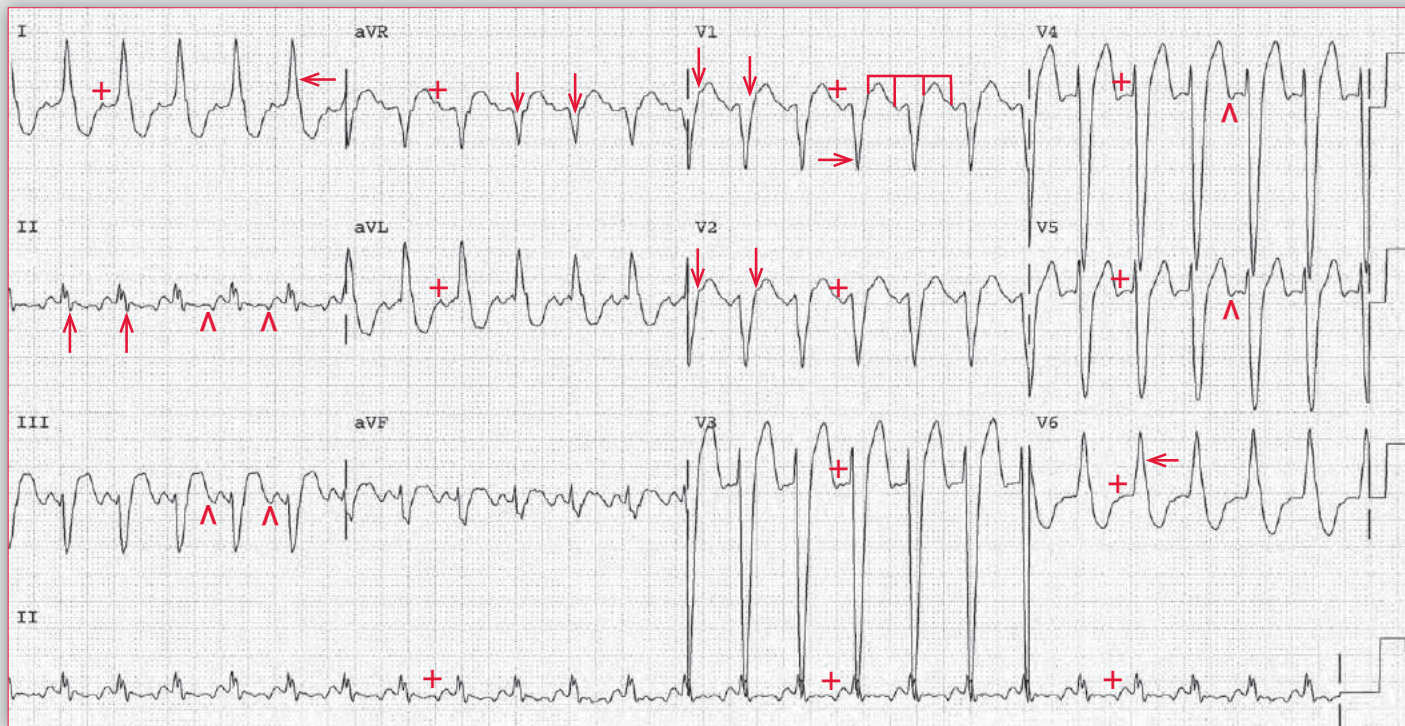
What do the ECGs show?

What is the likely precipitant for this particular episode of congestive heart failure?

ECG 68B



Podrid's Real-World ECGs



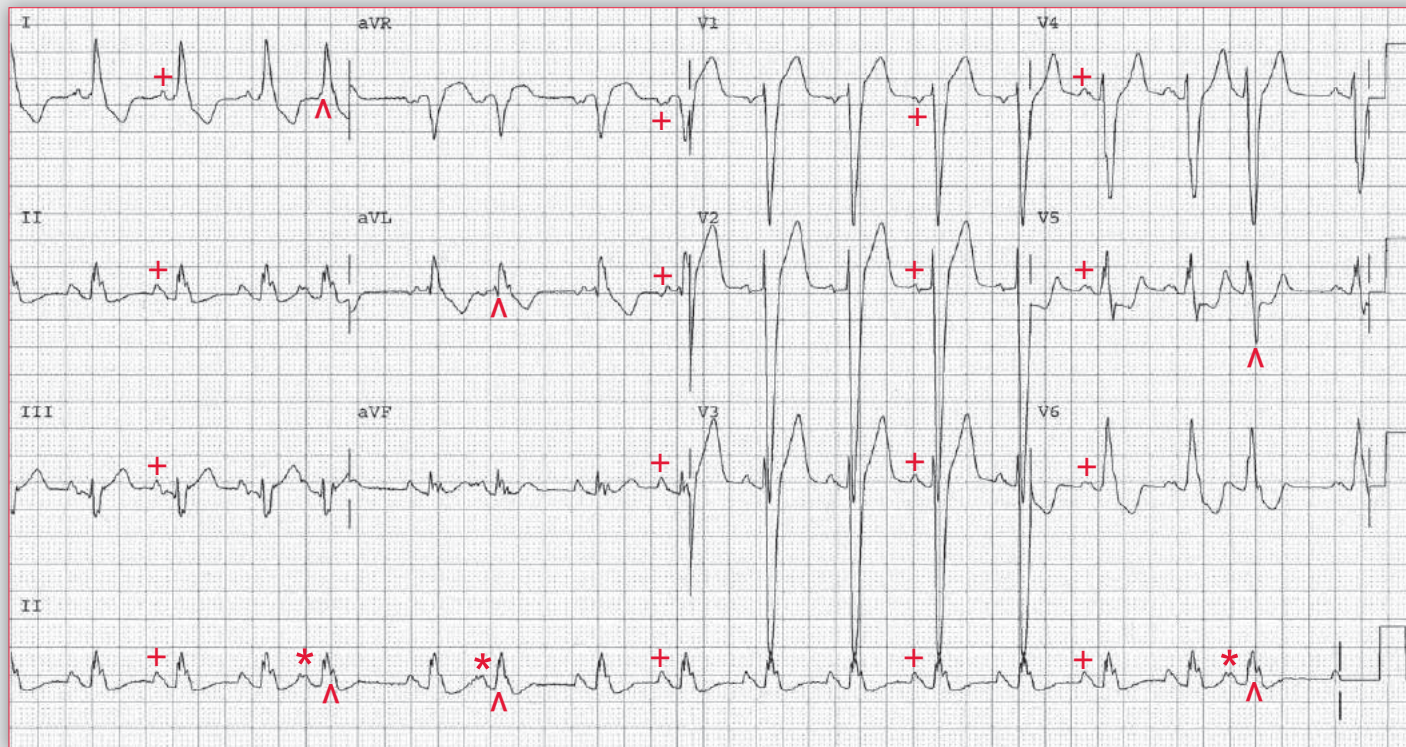
ECG 68A Analysis: Wide complex tachycardia, long PR tachycardia, atrial flutter with 2:1 AV block, left bundle branch block

ECG 68A shows a regular rhythm at a rate of 146 bpm. The QRS complex duration is prolonged (0.14 sec). The morphology is typical for a left bundle branch block, with a QS complex in lead V1 (→) and a broad R wave in leads I and V5–V6 (←). The axis is leftward between 0° and –30° with a positive QRS complex in leads I and II and a negative QRS in lead aVF. The QT/QTc intervals are normal (280/440 msec and 240/360 msec when the prolonged QRS complex duration is considered).

There is an atrial waveform seen before the QRS complexes (+) with a long RP (0.28 sec) and short PR (0.18 sec) interval. The P wave has an abnormal morphology, *ie*, negative–positive in leads II, III, aVF, and V4–V5 (^). There is a second atrial waveform, best seen in leads aVR and V1–V2 as a notching in the initial part of the ST segment (↓). This waveform can also be seen in lead II as a narrow terminal S wave (↑). The interval between these atrial waveforms is regular (□), at a rate of almost 300 bpm. Therefore, the rhythm is atrial flutter with 2:1 AV block.

continues

Podrid's Real-World ECGs



ECG 68B Analysis: Normal sinus rhythm, left atrial hypertrophy, left bundle branch block, and premature atrial complexes

ECG 68B is from the same patient as ECG 68A. The rhythm is regularly irregular, with 3 QRS complexes occurring early (^). The rest of the QRS complexes occur at a regular rhythm at a rate of 94 bpm. There is a P wave in front of these QRS complexes (+) with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. However, the P wave is very broad and notched, especially obvious in leads V3–V6. Therefore, there is left atrial hypertrophy or a left atrial abnormality.

The QRS complex duration and morphology (left bundle branch block) are the same as seen in ECG 68A. The QT/QTc intervals are also the same. The axis is, however, normal between 0° and +90° (positive QRS complex in leads I and aVF). Note that the notching of the ST segment seen in ECG 68A is not present, further supporting the fact that this was an atrial waveform and that the rhythm in ECG 68A is atrial flutter. The 3 premature complexes have the same QRS complex duration and morphology as the sinus complexes. There are P waves before the 3 premature complexes (*) with a PR interval of 0.22 second. These are premature atrial complexes. There is a fixed coupling interval between the preceding sinus complex and the premature complexes.

Atrial arrhythmias with rapid rates, including atrial flutter and atrial fibrillation, can often result in a worsening of heart failure in patients with a history of heart failure. This results from the rapid ventricular rate with a shortening of diastole, a reduction in left ventricular filling, and also the loss of effective atrial contraction, which may account for up to 30% to 40% of stroke volume in patients with underlying disease of the left ventricle. In addition, rapid supraventricular tachyarrhythmias that go unrecognized for some period of time (weeks or months) may result in the development of a tachycardia-mediated or -associated cardiomyopathy. This is due to multiple factors, including depletion of high-energy ATP stores and a decrease in the activity of the sodium-potassium ATPase pump, the occurrence of myocardial ischemia, abnormalities in myocardial blood flow, abnormal handling of calcium, down regulation of β receptors and reduction in β -receptor responsiveness, and oxidative stress with myocardial injury. The time course of the development of this cardiomyopathy is uncertain and likely highly variable. In many patients, there is improvement in left ventricular function when the rapid ventricular rate is controlled or the arrhythmia terminated. In many cases of a patient with normal left ventricular function prior to the arrhythmia, left ventricular function normalizes. ■

Practice Case 69

A 30-year-old man presents to the emergency department with palpitations and mild dyspnea. Physical examination is unremarkable except to the finding of a rapid heart rate and a blood pressure of 80/60 mm Hg. An ECG is obtained (ECG 69A).

As it was felt that the patient was hemodynamically compromised, electrocardioversion was performed in the emergency department.

A subsequent ECG was obtained (ECG 69A).

ECG 69A

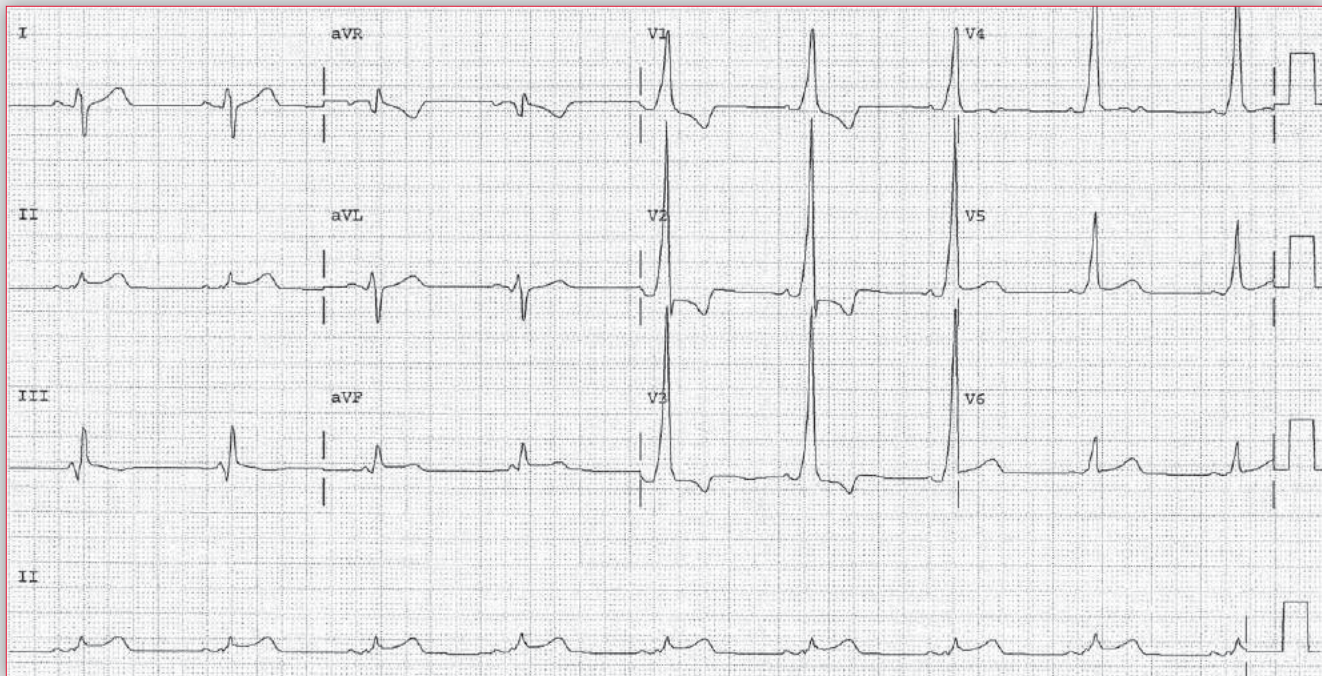


Practice Case 69

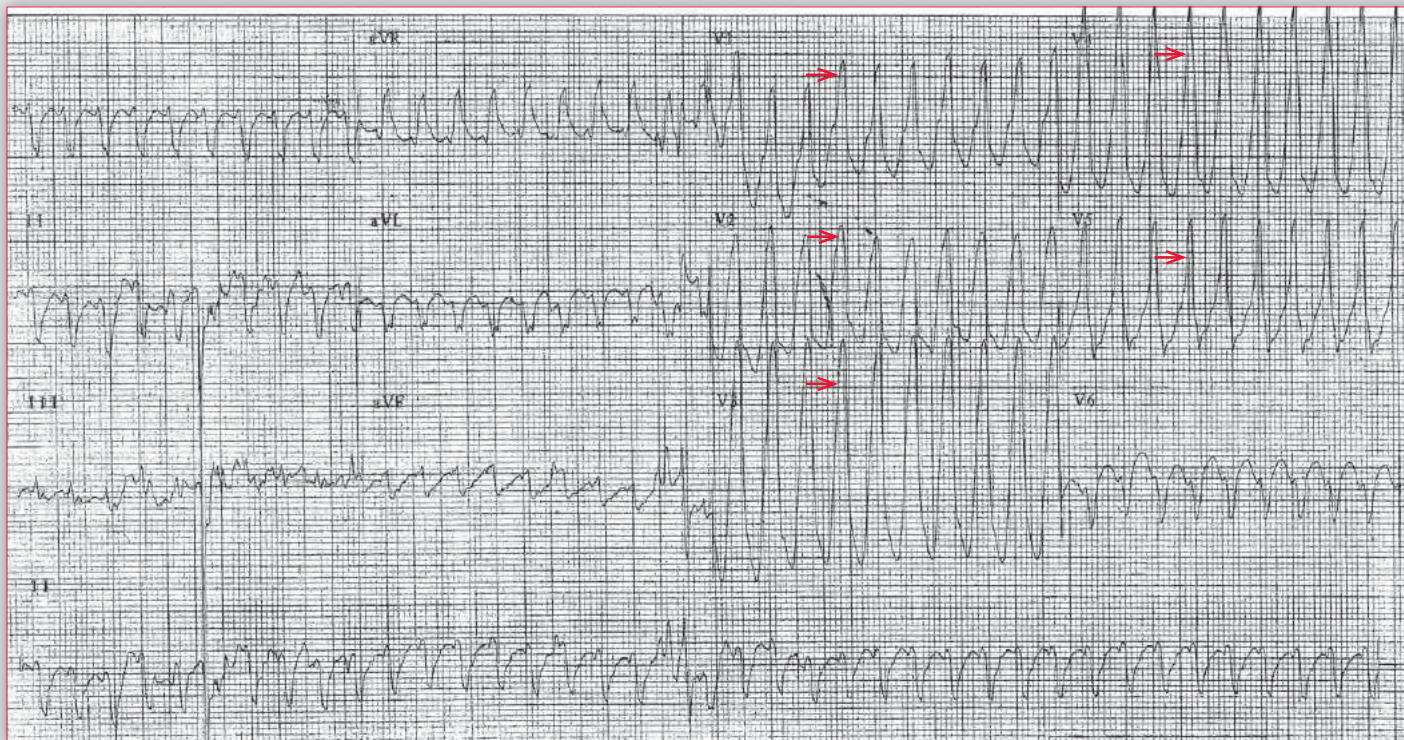
What is the differential diagnosis for the tachycardia seen in ECG 69A?

Using the two ECGs together, what diagnosis is now established?

ECG 69B



Podrid's Real-World ECGs



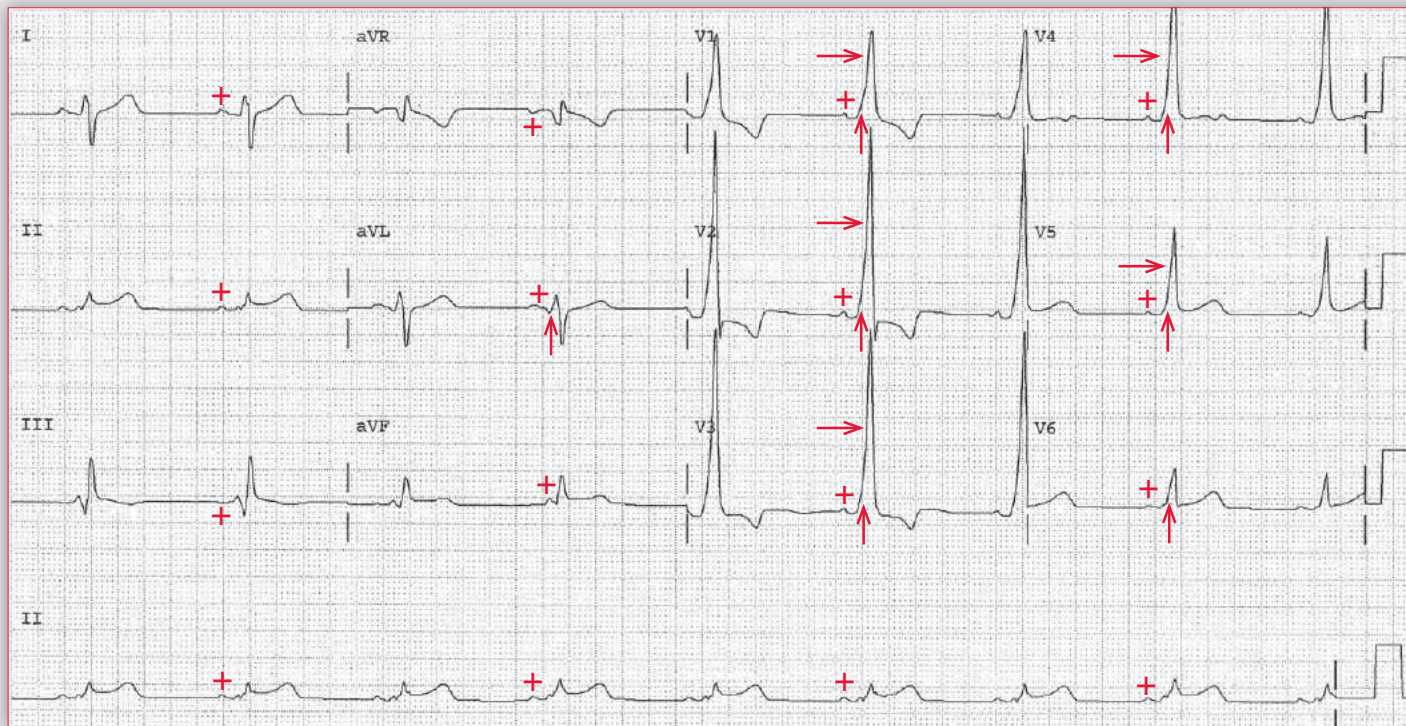
ECG 69A Analysis: Wide complex tachycardia at 240 bpm, right bundle branch-like morphology, right axis, antidromic atrioventricular reentrant tachycardia

ECG 69A shows a regular rhythm at a rate of 240 bpm. There are no obvious P waves seen. The QRS complex duration is increased (0.16 sec). Although the QRS complex morphology resembles a right bundle branch block, it is not typical and is abnormal, as there are tall, broad R waves in V1–V5 (→) (almost positive concordance across the precordium), which is not a typical pattern for a right bundle branch block. The axis is rightward between $+90^\circ$ and $+180^\circ$ (negative QRS complex in lead I and positive in lead aVF). The QRS complexes and

ST-T waves appear to be uniform in morphology. In view of the very abnormal QRS complex morphology, the differential is either a ventricular tachycardia or a supraventricular tachycardia with aberration with an unusual QRS morphology, specifically an atrioventricular reentrant tachycardia (AVRT) associated with Wolff-Parkinson-White. As the QRS complexes are wide and abnormal, this would be an antidromic AVRT. However, there are no specific findings on this ECG that would be useful for establishing the actual etiology.

continues

Podrid's Real-World ECGs



ECG 69B Analysis: Sinus bradycardia, Wolff-Parkinson-White pattern

ECG 69B is from the same patient as ECG 69A and was obtained after cardioversion. There is a regular rhythm at a rate of 52 bpm. There is a P wave before each QRS complex (+) with a stable but short PR interval (0.12 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus bradycardia. The QRS complex duration is increased (0.16 sec) and there is positive concordance across the precordium (\rightarrow) (*ie*, tall R waves in leads V1–V6). The axis is rightward between $+90^\circ$ and $+180^\circ$ (negative QRS complex in lead I and positive in lead aVF). Noted in most leads is prominent slurring of the initial upstroke of the QRS complex (\uparrow). This initial slurring indicates that there is direct initial activation of the ventricular myocardium, which is slow. This can occur with a ventricular complex, a paced complex, or with Wolff-Parkinson-White pattern in which the initial ventricular activation is via an accessory pathway. In this situation, the slurred upstroke is called a delta wave. The finding of this slurred upstroke along with a short PR interval is diagnostic of a Wolff-Parkinson-White pattern.

Comparison with the QRS complexes seen during the tachycardia in ECG 69A demonstrates that the QRS morphology is the same, which supports the diagnosis of an AVRT that is antidromic, *ie*, the impulse travels from the atrium antegradely via the accessory pathway to activate the ventricle, while the retrograde limb of the circuit that results in VA conduction back to the atrium is via the AV node–His-Purkinje system. As ventricular activation is direct via the accessory pathway, the QRS complexes will be preexcited and have the same WPW morphology as those seen during sinus rhythm, although the complexes

will be wider as they will be maximally preexcited, *ie*, all of ventricular activation occurs via the accessory pathway as opposed to the sinus complexes, which are a fusion between accessory pathway activation and activation via the AV node–His-Purkinje system. Since antidromic AVRT is the result of direct ventricular activation and can be similar to ventricular tachycardia, establishing this arrhythmia as the etiology may be difficult. However, it is of great importance in regard to treatment, as an AV nodal blocking agent that might terminate an AVRT will potentially worsen ventricular tachycardia and provoke ventricular fibrillation. The important issue in diagnosing an antidromic AVRT is comparison with the preexcited complex during sinus rhythm. QRS complexes during the tachycardia that are identical to those of the preexcited complexes in sinus rhythm, although often wider as the complexes are more preexcited, is supportive evidence for an antidromic AVRT. Other findings of importance for distinguishing between these two arrhythmias is AV dissociation, which is the hallmark of ventricular tachycardia and cannot be seen with an AVRT, as there needs to be an intact circuit for this arrhythmia to occur, and that means that there must be 1:1 AV conduction. Also of important for establishing ventricular tachycardia is the presence of variability of the QRS complexes and ST-T waves, which cannot be seen with AVRT as there is a fixed circuit and hence every QRS complex and ST-T wave must be identical.

If an antidromic AVRT is established as the etiology for the tachycardia, treatment is identical to that in orthodromic AVRT, *ie*, interruption of the circuit will terminate the arrhythmia. Hence any AV nodal blocking agent can be used. ■

Practice Case 70

A 71-year-old woman becomes short of breath and very lightheaded while shopping. Emergency personnel are called, and when they arrive on the scene, they obtain an ECG (ECG 70A). She is brought to an emergency department and a few minutes later, her heart rate has slowed (ECG 70B). She is alert but confused and mildly dyspneic.

ECG 70A

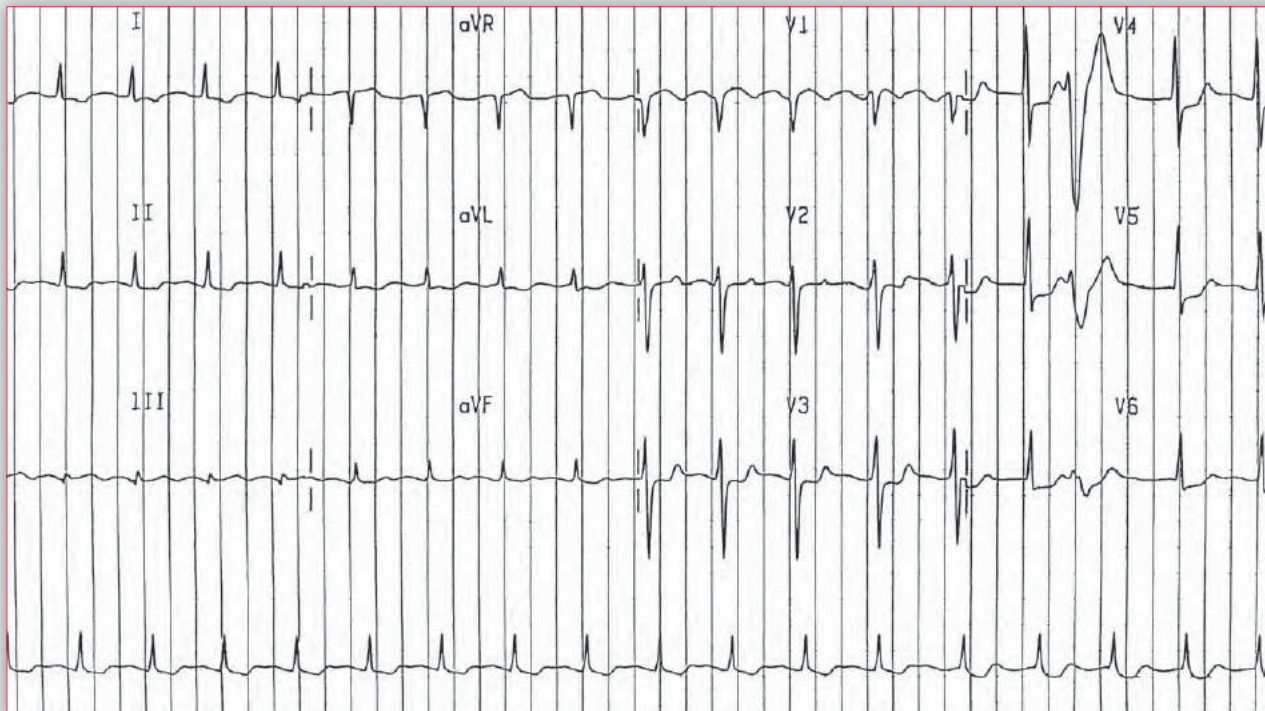


Practice Case 70

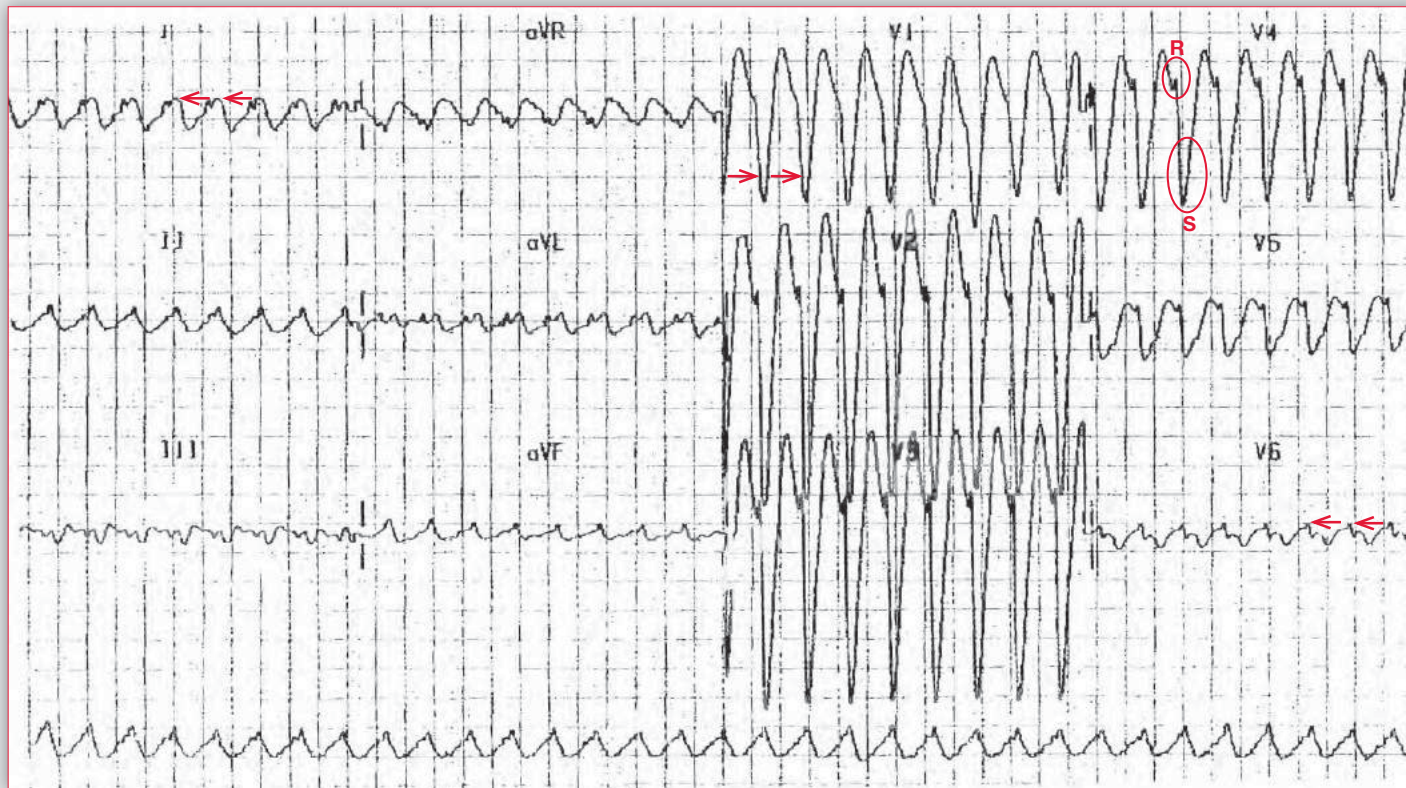
What do the ECGs show?

What is the underlying diagnosis?

ECG 70B



Podrid's Real-World ECGs



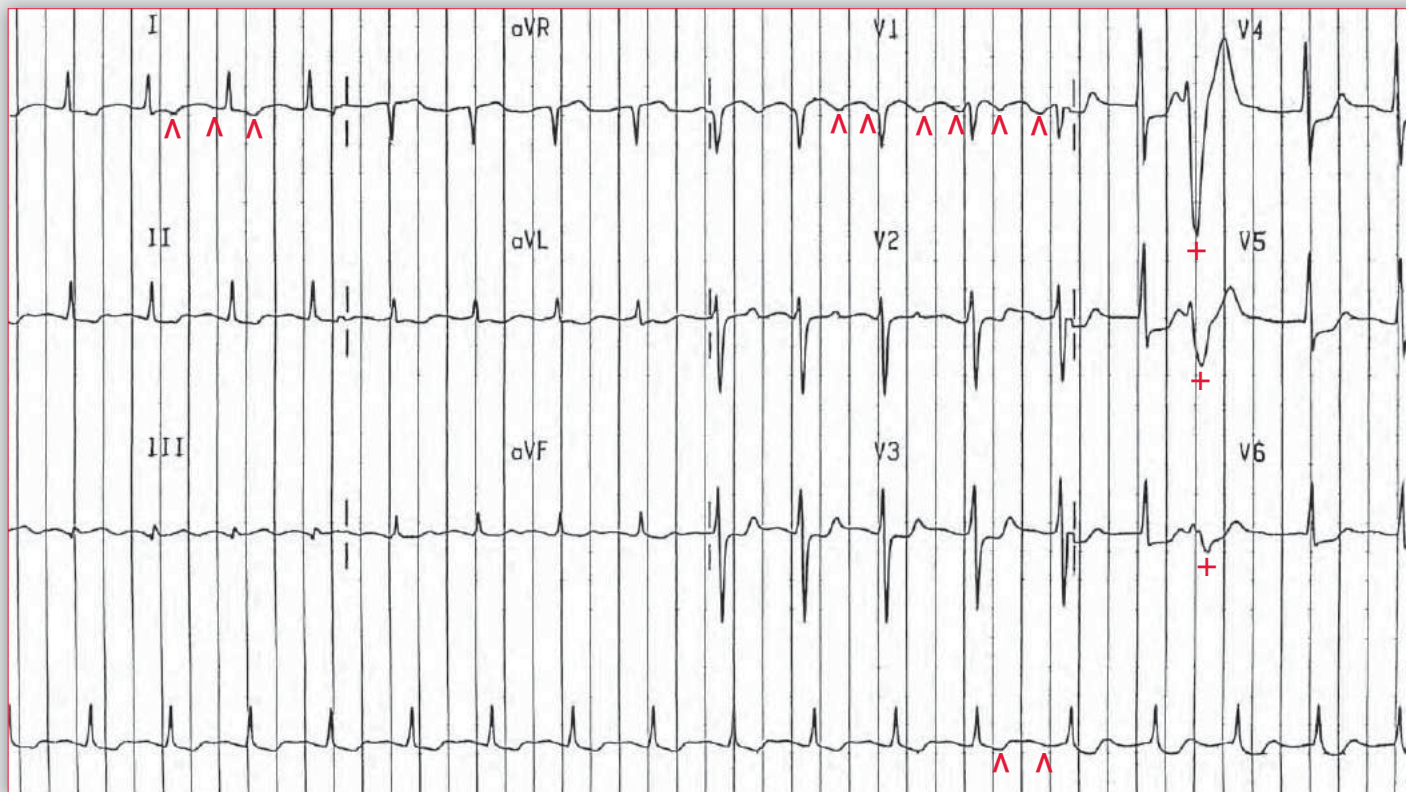
ECG 70A Analysis: Wide complex tachycardia, left bundle branch block (rate-related), atrial flutter with 1:1 conduction

ECG 70A shows a regular rhythm at a rate of 250 bpm. The QRS complex duration is increased (0.16 sec), and there is a typical left bundle branch block pattern present with a deep QS complex in lead V1 (→) and tall R waves in leads I and V6 (←). There are no obvious P waves seen. The two rhythms occurring at this rate are ventricular tachycardia, which at this rate is termed ventricular flutter, or atrial flutter with 1:1 AV conduction. The diagnosis is more likely to be a

supraventricular tachyarrhythmia, *ie*, atrial flutter, based on the fact that there is a typical left bundle branch block pattern present. In addition, there is an R/S complex present in leads V2–V4. The R wave is narrower (< 0.08 msec) than the S wave. This is consistent with a supraventricular rhythm with a left bundle branch block aberration as the initial forces are normal, while it is the terminal portion that is aberrated and responsible for the widened QRS complex, *ie*, a bundle branch block.

continues

Podrid's Real-World ECGs



ECG 70B Analysis: Atrial flutter with 2:1 AV block and one episode of 1:1 AV conduction with rate-related left bundle branch block

ECG 70B is from the same patient as ECG 70A. There is regular rhythm at a rate of 125 bpm. It should be noted that the rhythm strip is not simultaneous with the 12-lead ECG. The QRS complex duration is normal (0.08 sec). The QRS morphology is normal and the axis is normal between 0° and $+90^{\circ}$. The QT/QTc intervals are normal (300/430 msec).

Although there are no distinct P waves seen, there is evidence of atrial activity (^), particularly in leads I and V1. The atrial activity is regular with an undulating (up and down) pattern. The atrial rate is 250 bpm, which is identical to the ventricular rate seen in ECG 70A. These are atrial flutter waveforms, although the rate is slightly slower than what is typical for atrial flutter (*ie*, 260 to 320 bpm). However, there is a

typical flutter morphology with continuous undulation of the atrial waveforms without an isoelectric baseline between each waveform. Hence, this is atrial flutter with 2:1 AV conduction. Note that a left bundle branch block pattern is not seen and, therefore, there was a rate-related left bundle branch block seen in ECG 70A. Importantly, the fifteenth QRS complex (+) is early and has a rate (RR interval) that is almost the same as the RR intervals or rate seen in ECG 70A as well as the atrial flutter rate. Therefore, this is a transient episode of 1:1 AV conduction. The QRS complex is wide and has a left bundle branch block morphology that is identical to the QRS complexes seen in ECG 70A. Therefore, this confirms the presence of a rate-related left bundle branch block. ■

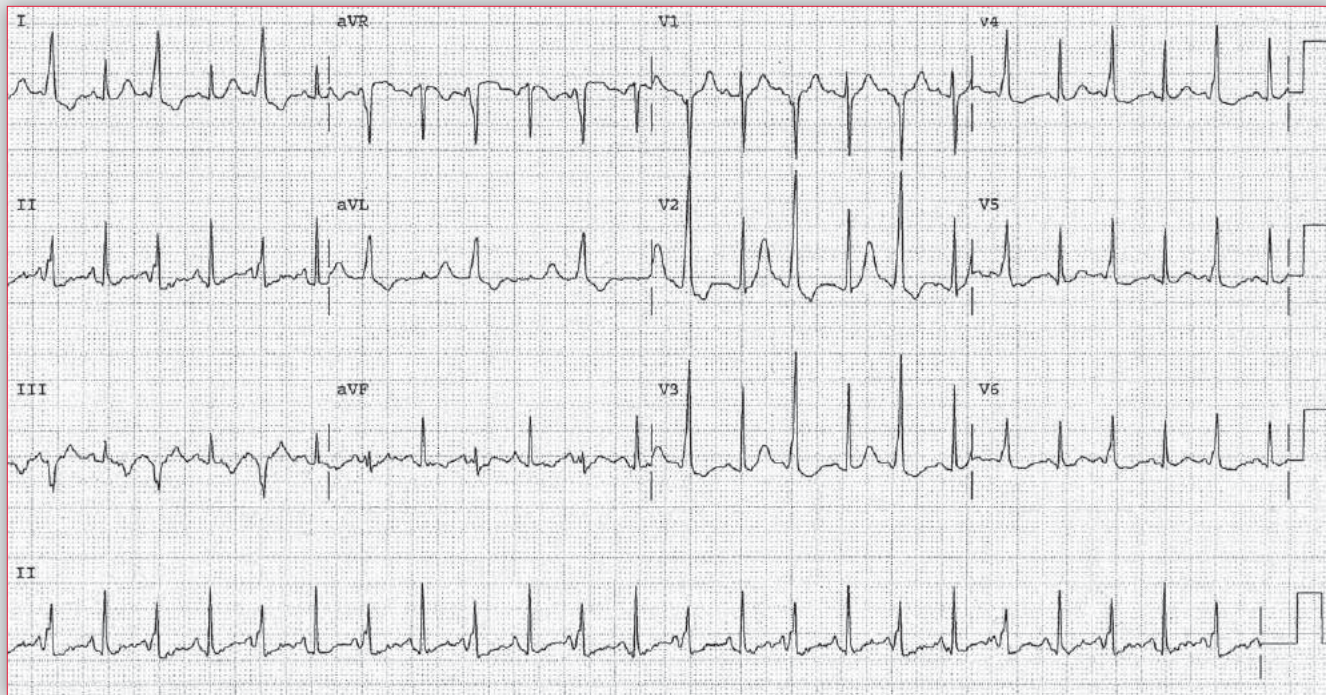
Notes

Practice Case 71

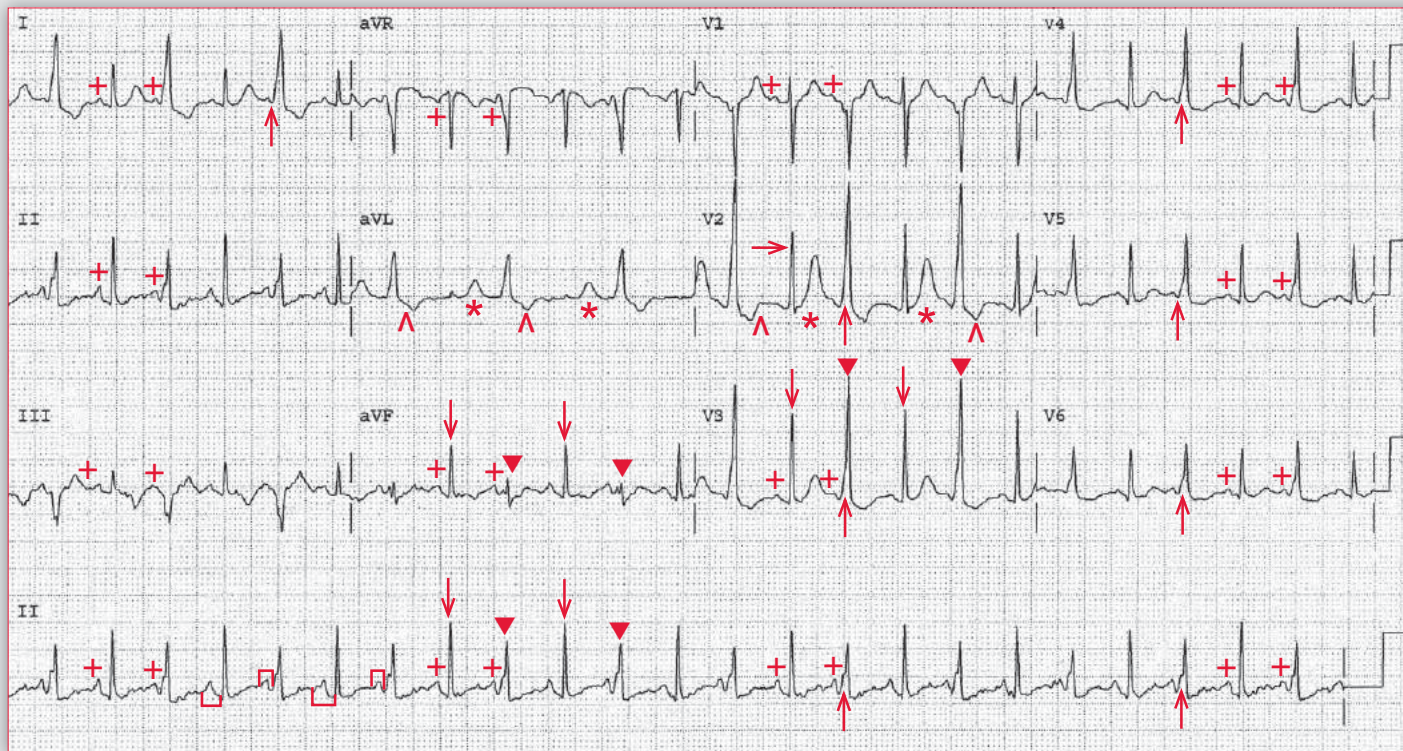
An 18-year-old woman has an episode of tachycardia and pre-syncope while playing soccer. She is taken off the field and when EMS arrives she is feeling better. An ECG is obtained.

What does the ECG show?

What is the underlying diagnosis?



Podrid's Real-World ECGs



ECG 71 Analysis: Sinus tachycardia with intermittent Wolff-Parkinson-White pattern

ECG 71 shows a regular rhythm at a rate of 140 bpm. There is a P wave (+) before each QRS complex and the PP intervals are constant. The P wave is positive in leads I, II, aVF, and V4–V6. Hence there is a sinus tachycardia. There are beat-to-beat changes in the PR interval (\square , \sqcup) as well as QRS complex duration and amplitude (\downarrow , \blacktriangledown), suggesting electrical alternans. The narrow QRS complexes (\downarrow) have a duration of 0.08 second and a PR interval of 0.12 second (\sqcup). The wide QRS complexes (\blacktriangledown) have a duration of 0.12 second and PR interval (\square) of 0.08 second. The narrow QRS complexes have a normal axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF) and morphology with early transition, *ie*, tall R wave in lead V2 (\rightarrow). This is termed counterclockwise rotation of the electrical axis in the horizontal plane and it is determined by imagining the heart as being looked at from under the diaphragm. With counterclockwise rotation, the left ventricular forces occur earlier in the precordium; hence the tall R wave in V2.

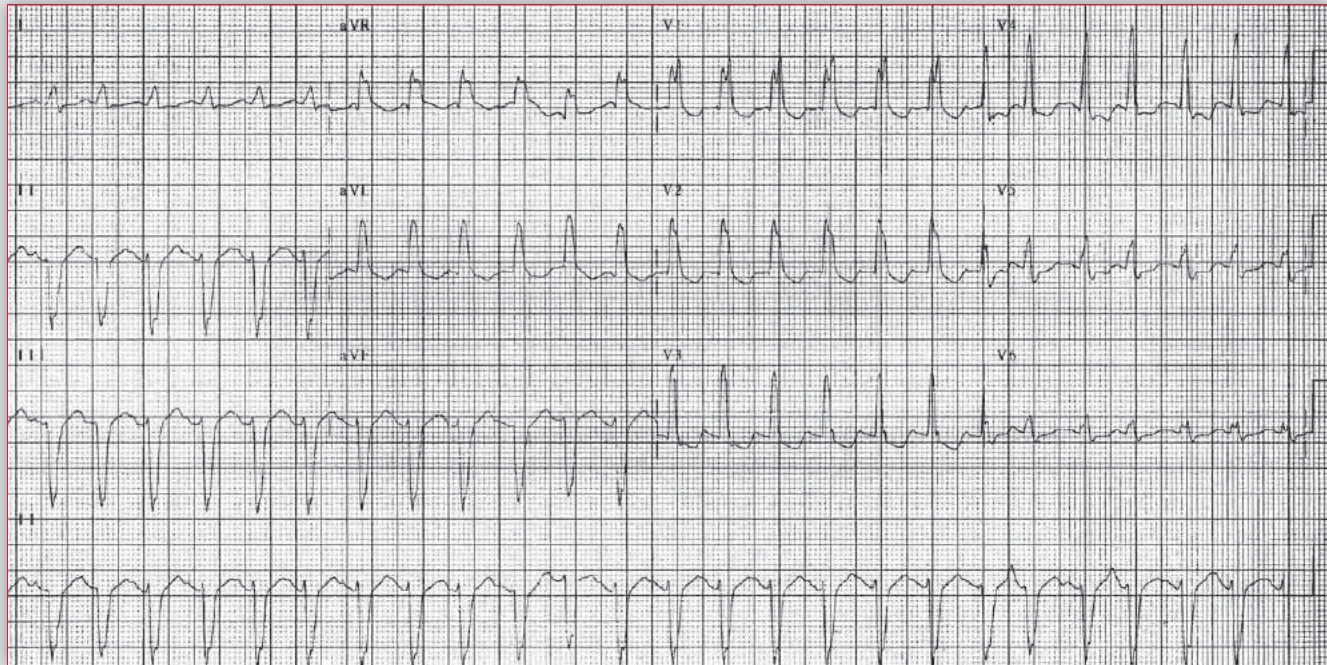
The wide QRS complexes have a slurred upstroke (\uparrow), best seen in leads V2–V6. Along with the short PR interval, this represents a Wolff-Parkinson-White (WPW) pattern. Therefore, this is intermittent (beat-to-beat) WPW pattern. Although it looks like electrical or QRS alternans as well as T-wave alternans ($*$, \wedge), the QRS complexes have beat-to-beat changes in width and morphology, *ie*, preexcited and nonpreexcited, which accounts for the beat-to-beat changes in QRS amplitude and T-wave morphology.

The occurrence of intermittent WPW suggests that the refractoriness of the accessory pathway is relatively long; hence it is not capable of conducting impulses at a rapid rate. It is likely that this pattern is seen in this case as the result of the sinus tachycardia. In general, patients who manifest intermittent WPW pattern do not have an increased risk for sudden death, which is usually a result of atrial fibrillation resulting in a very rapid ventricular response rate (often > 320 bpm) due to the ability of the accessory pathway to conduct impulses at a rapid rate. The very rapid ventricular rate can precipitate ventricular fibrillation. ■

Practice Case 72

An 80-year-old woman with moderate degenerative aortic stenosis presents with worsening dyspnea. An ECG (ECG 72A) is obtained, and as a result she was treated with adenosine. However, there was no response. A cardiology consult was called to provide further options for therapy. The ECG was compared to the patient's baseline ECG (ECG 72B).

ECG 72A



Practice Case 72

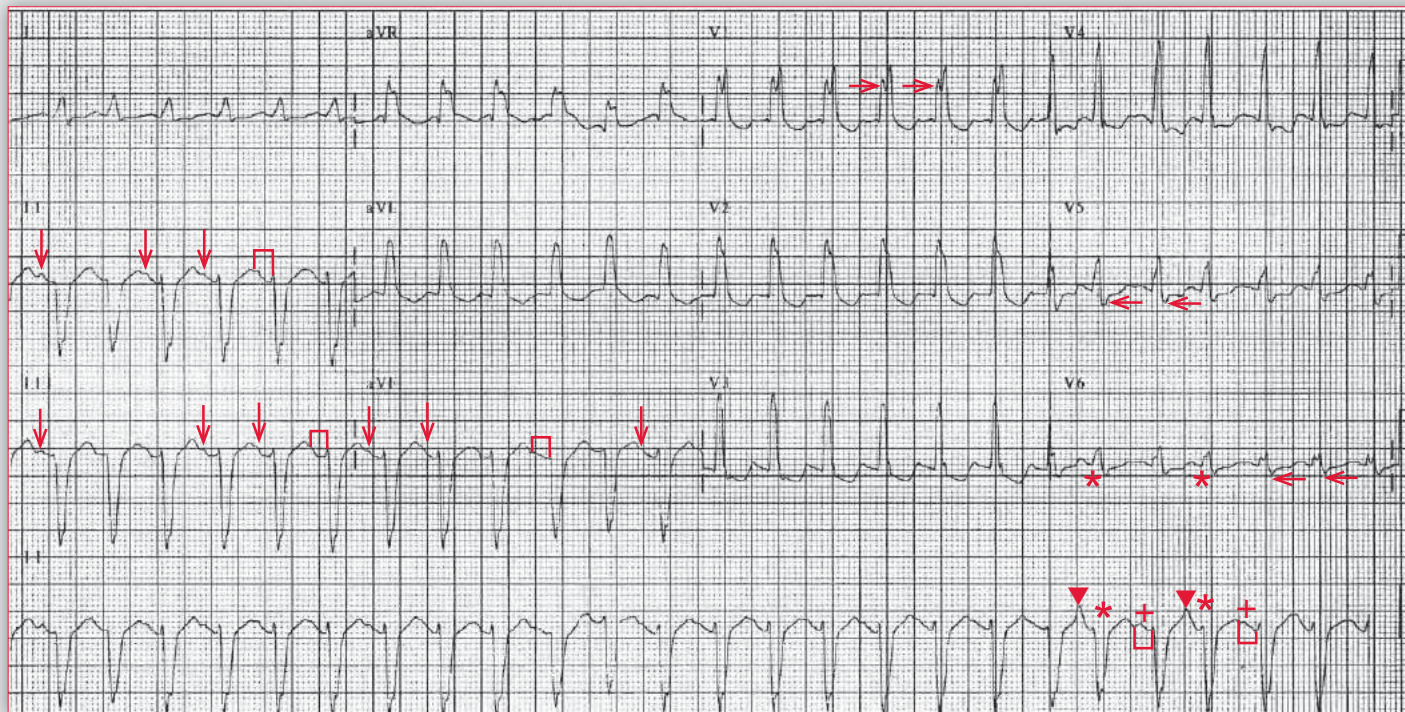
What does the ECG show?

Is any additional therapy necessary?

ECG 72B



Podrid's Real-World ECGs



ECG 72A Analysis: Sinus tachycardia, right bundle branch block, left anterior fascicular block, premature atrial complexes

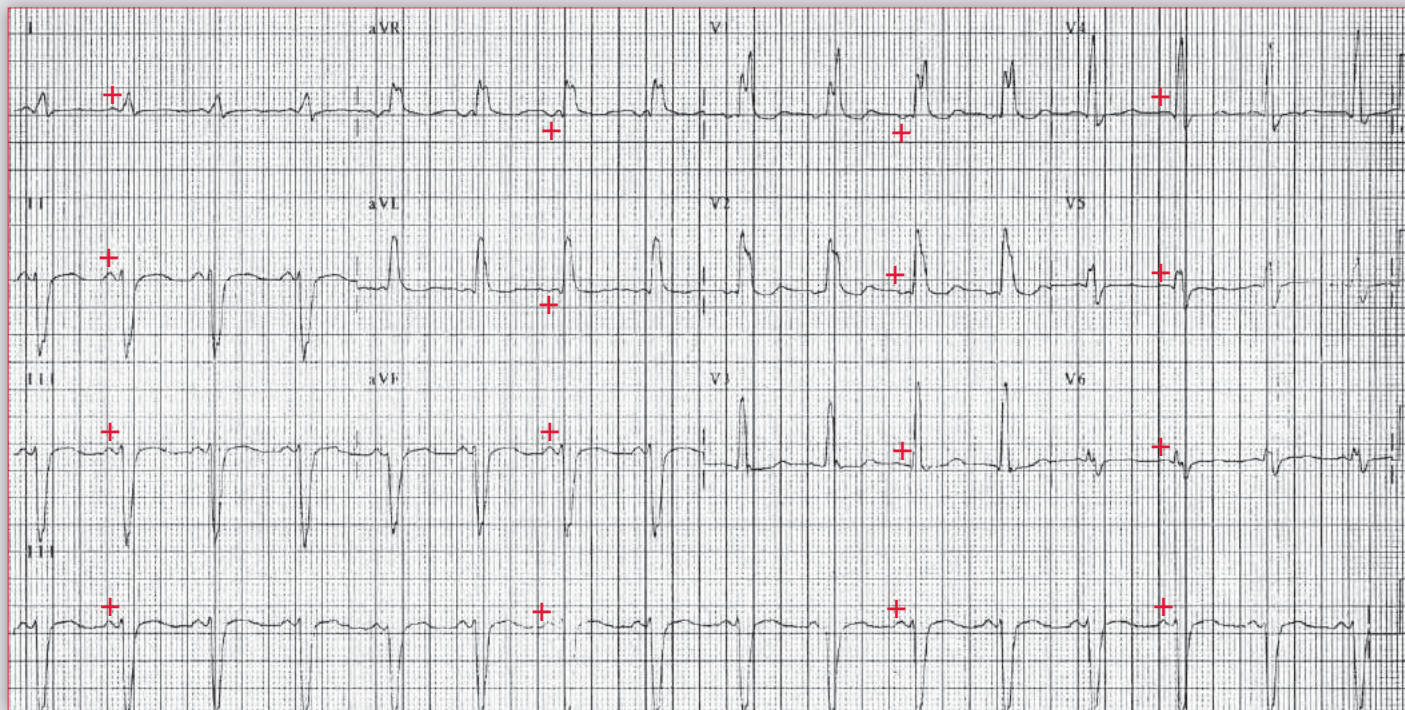
ECG 72A shows a regular rhythm at a rate of 148 bpm. Although P waves are not immediately obvious, careful inspection shows that there are waveforms that look like atrial activity. They are best seen at the end of the T wave in leads II, III, and aVF (↓). There is a long RP interval (0.20 sec) and a short PR interval (0.14 sec). When the ECG was first recorded, the rhythm is a long RP tachycardia. There are a number of etiologies, including sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, typical atrioventricular nodal reentrant tachycardia of an unusual variant (slows-slow), or an atrioventricular reentrant tachycardia. In addition, there are two premature complexes (twentieth and twenty-second) (*), after which there is a pause. During the pause, a clear P wave can be seen (+), with a stable PR interval of 0.14 second (□). Using this PR interval as the baseline, it can be seen that waveforms noted on the T wave in leads II, III, and aVF are indeed P waves with a stable PR interval (□). The P wave is upright in leads II and aVF and, therefore, this is a sinus tachycardia and not an arrhythmia.

The QRS complex duration is prolonged (0.12 sec) and there is a morphology that looks like a right bundle branch block with a broad RR' in lead V1 (→) and broad S wave in leads V5–V6 (←). There is also an extreme leftward axis between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF with an rS complex). This is a left anterior fascicular block. The QT/QTc intervals are normal (280/440 msec and 260/400 msec when corrected for the prolonged QRS complex duration).

The two premature complexes have the same QRS complex morphology as the sinus complexes. Although there is no obvious P wave before these two complexes, noted is a major change in the T-wave amplitude (▼), which is the result of a superimposed P wave. Hence these are premature atrial complexes.

continues

Podrid's Real-World ECGs



ECG 72B Analysis: Normal sinus rhythm with right bundle branch block,
left anterior fascicular block

ECG 72B is from the same patient as ECG 72A and shows a regular rhythm at a rate of 90 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.14 sec), the same as the PR interval seen in ECG 72A. In addition, the QRS duration, morphology, and axis are identical to the QRS complexes in ECG 72A. Hence ECG 72A shows a sinus tachycardia and premature atrial complexes with a right bundle branch block and left anterior fascicular block; the conduction abnormalities are preexistent, being present during sinus rhythm. Hence the QRS complex abnormality in ECG 72A is not rate-related, but is the baseline complex.

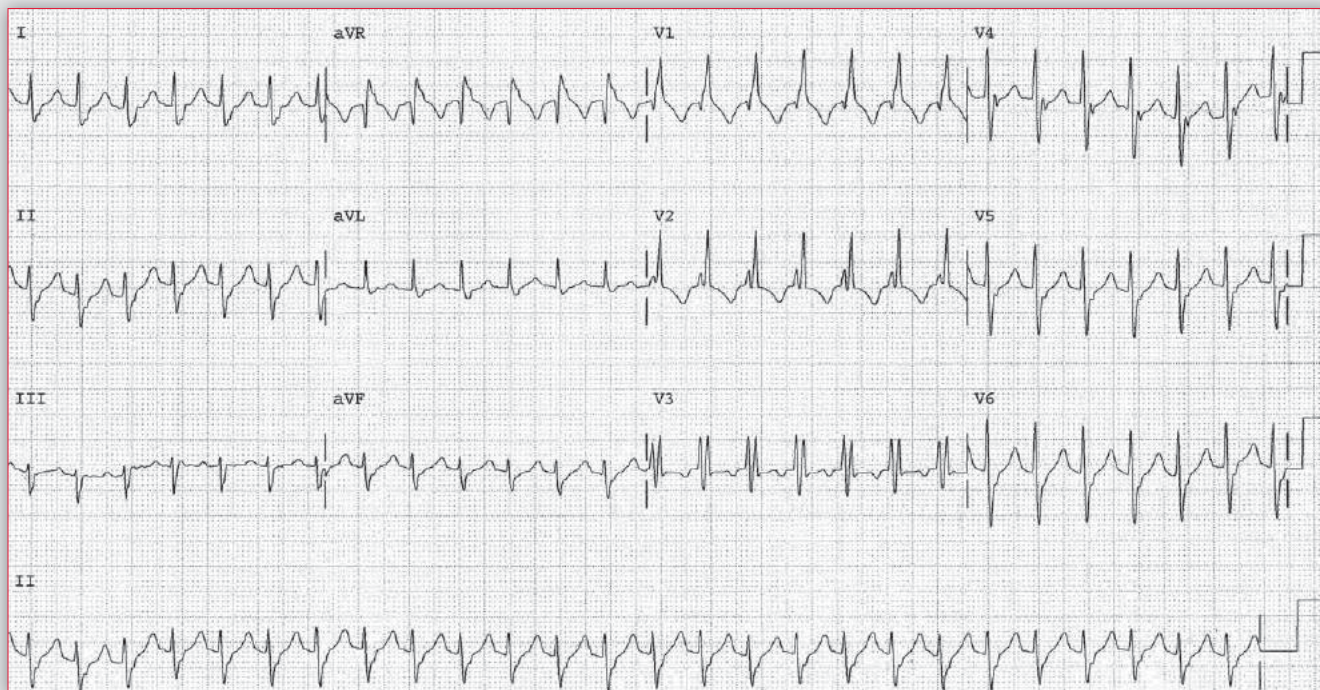
It is important that, in the presence of a tachycardia, a P wave be looked for superimposed on T waves, especially if there is a prolonged PR interval. Hence sinus tachycardia may be associated with notches,

bumps, or irregularities of the T waves, which should have a smooth upstroke and downstroke. In addition, P waves should be looked for after a pause or long RR interval. If the P wave is seen, this will establish the PR interval, which will be helpful for the identification of other P waves, particularly if they are superimposed on T waves. The initial diagnosis for the arrhythmia was considered to be an atrioventricular nodal reentrant tachycardia, as P waves were not obvious. It was for this reason that adenosine was administered. Since the arrhythmia was not reentrant involving the AV node, this agent was not effective. Sinus tachycardia is most often physiologic and is the result of sympathetic stimulation or an increase of circulation catecholamines. Hence adenosine will not change the sinus rate, although there is the potential for AV block. ■

Practice Case 73

A 44-year-old man has longstanding palpitations that are relatively brief, lasting for less than one hour. No etiology has ever been established because an ECG has never been recorded during an event, as the arrhythmia terminates before he gets to an emergency department. However, he has an episode that is more prolonged, and in the emergency department an ECG is obtained during the episode of the arrhythmia.

ECG 73A

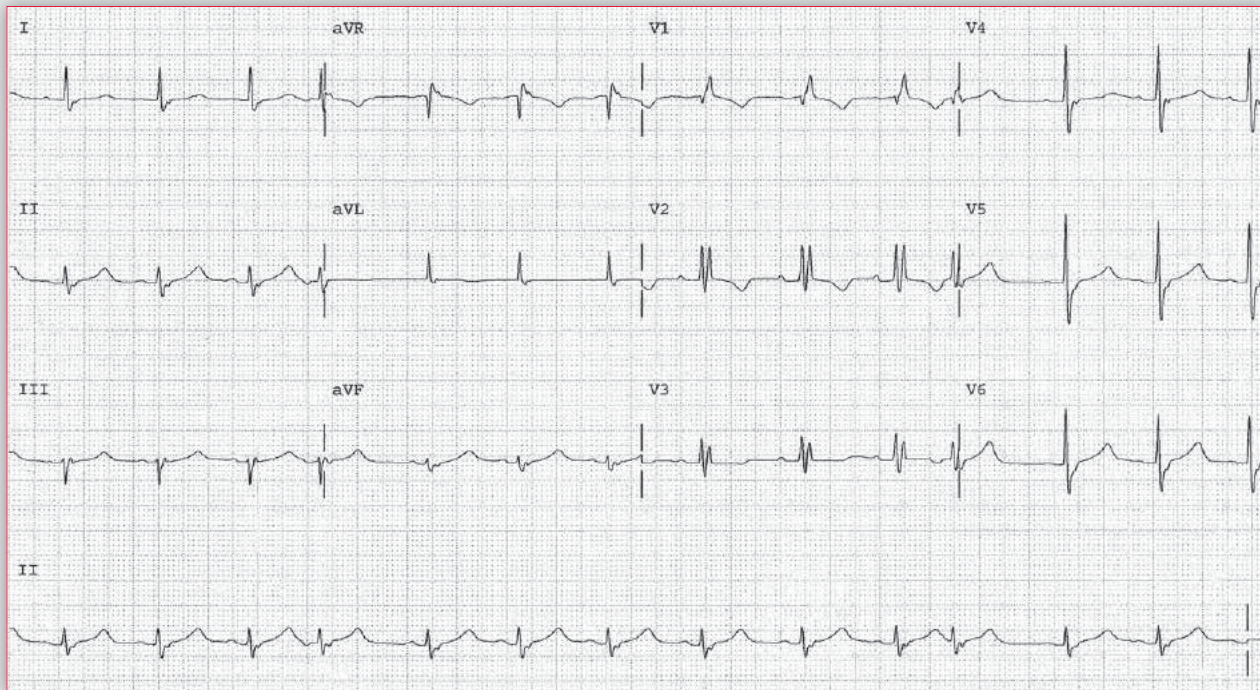


Practice Case 73

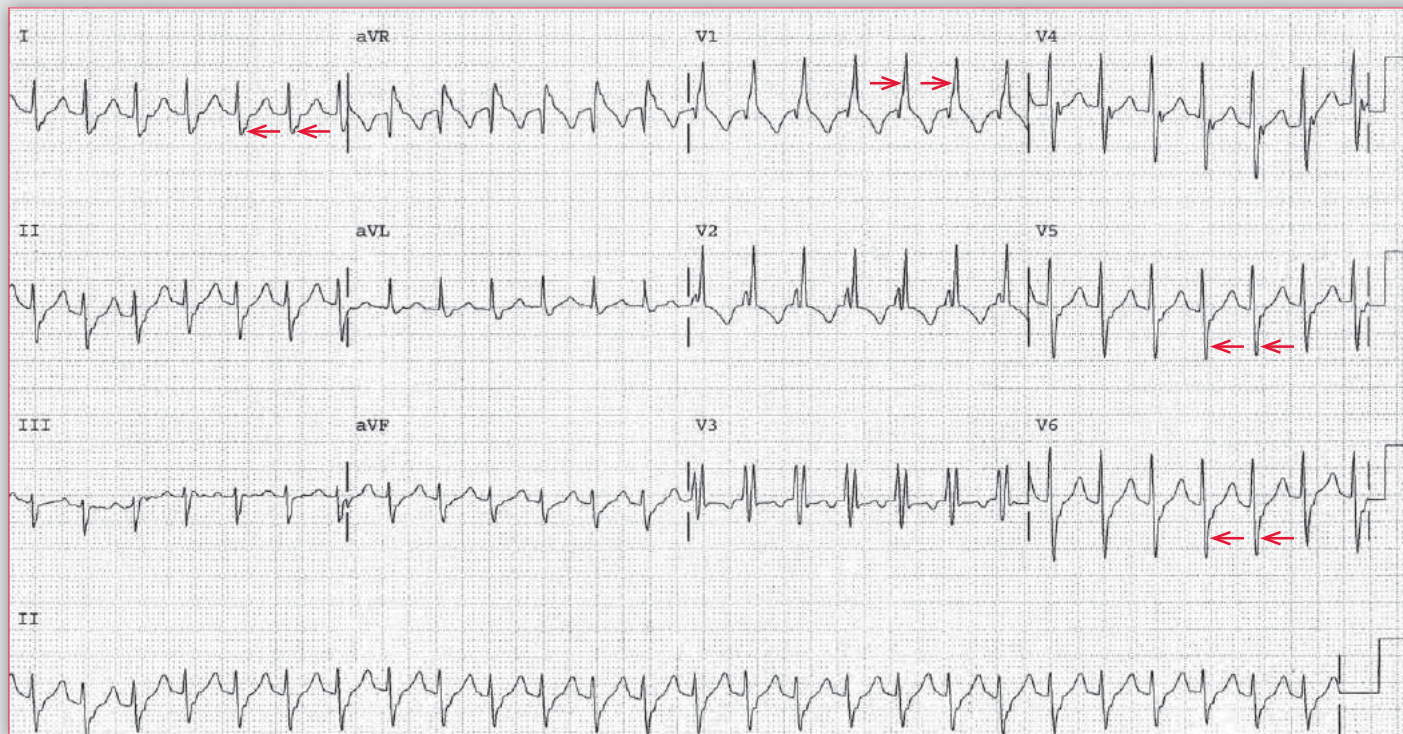
What is his likely diagnosis?

What therapies will be effective for treatment?

ECG 73B



Podrid's Real-World ECGs



ECG 73A Analysis: Narrow complex supraventricular tachycardia (no-RP tachycardia), is atrioventricular nodal reentrant tachycardia, right bundle branch block morphology, left anterior fascicular block

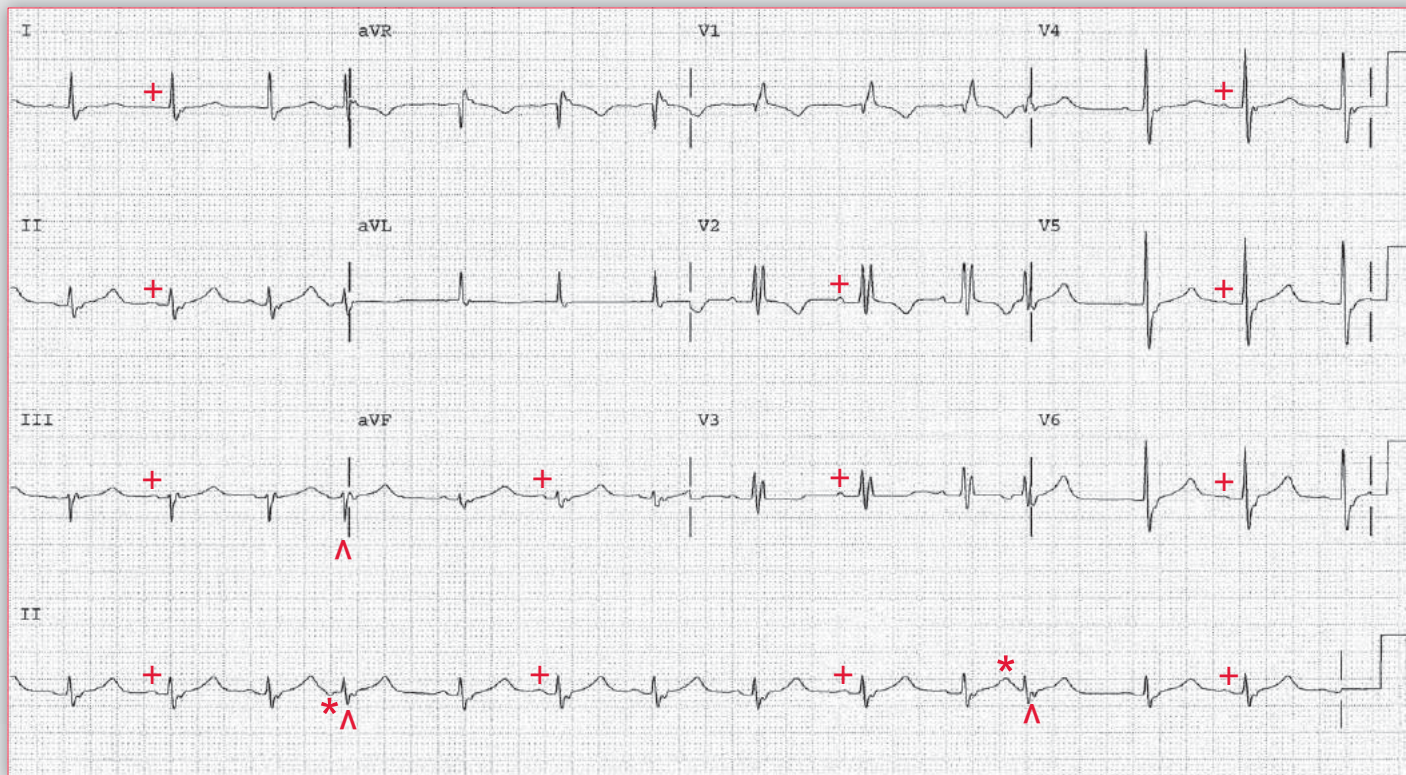
ECG 73A shows a regular rhythm at a rate of 150 bpm. The QRS complex duration is prolonged (0.12 sec) and it has a typical right bundle branch block morphology with an RSR' in lead V1 (→) and a terminal S wave in leads I and V5–V6 (←). The axis is extremely leftward between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF with an rS morphology). This is a left anterior fascicular block. The presence of a right bundle branch block and left anterior fascicular block is termed bifascicular disease. The QT/QTc intervals are prolonged (300/470 msec), but are normal when the prolonged QRS complex duration is considered (280/430 msec).

There are no P waves seen before or after any of the QRS complexes. Hence this might be termed a no-RP tachycardia, and the most common etiology for this is a typical or common atrioventricular nodal reentrant tachycardia (AVNRT). Uncommonly, this may be an atrial tachycardia or an ectopic junctional tachycardia. Atypical AVNRT is

due to the presence of dual AV nodal pathways, *ie*, a slow pathway that conducts slowly but has a short refractory period and hence recovers quickly, and a fast pathway that conducts rapidly but has a long refractory period and hence recovers slowly. These pathways are linked proximally and distally in the AV node. When there is a premature atrial complex that occurs before the fast pathway has recovered, the impulse will be conducted antegradely to the ventricles via the slow pathway. If the impulse reaches the distal end of the circuit at a time when the fast pathway has recovered, the impulse enters this pathway to retrogradely activate the atrium. If the impulse reaches the proximal portion of the circuit when the slow pathway has recovered, the impulse reenters the slow pathway. If this process continues, a reentrant arrhythmia is established. No P wave is seen because there is simultaneous activation of the atria in a retrograde direction via the fast pathway and antegrade activation of the ventricles via the slow pathway.

continues

Podrid's Real-World ECGs



ECG 73B Analysis: Normal sinus rhythm, right bundle branch block, left anterior fascicular block

ECG 73B is from the same patient as ECG 73A. There is a regular rhythm at a rate of 76 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration and morphology, and axis are identical to what is seen in ECG 73A. However, the axis is no longer extremely leftward (*ie*, it is between 0° and –30°, as the QRS complex in lead II is positive. Hence a left anterior fascicular block is no longer present and was probably rate-related. By comparing the complexes with those in ECG 73A, there is no evidence for any atrial activity in ECG 73A. The QT/QTc intervals are also the same. Hence there is an underlying right bundle branch block and a left anterior fascicular block.

The fourth and eleventh QRS complexes are early (^) and they are preceded by a P wave (*) that is different from the sinus P waves. The QRS complex has the same morphology as the sinus complexes. Hence

these are premature atrial complexes. The P waves and PR intervals in front of these two premature atrial complexes are different from each other; thus these are multifocal premature atrial complexes.

As atypical AVRNT is due to reentry within the AV node, any drug that alters the electrophysiologic properties of AV nodal conduction or repolarization can terminate this arrhythmia acutely and prevent its occurrence long term. Intravenous adenosine is the most common agent given for acute termination, as it is very effective and has a very short half-life of < 20 seconds. Other agents of benefit for acute termination include β -blockers, calcium-channel blockers (verapamil or diltiazem), or digoxin. Vagal maneuvers such as carotid sinus pressure or Valsalva may also be useful for acute termination of the arrhythmia. Long-term therapy involves AV nodal blocking agents (β -blockers, calcium-channel blockers, or digoxin); class IA, IC, or III antiarrhythmic drugs; or radiofrequency ablation of the AV node. ■

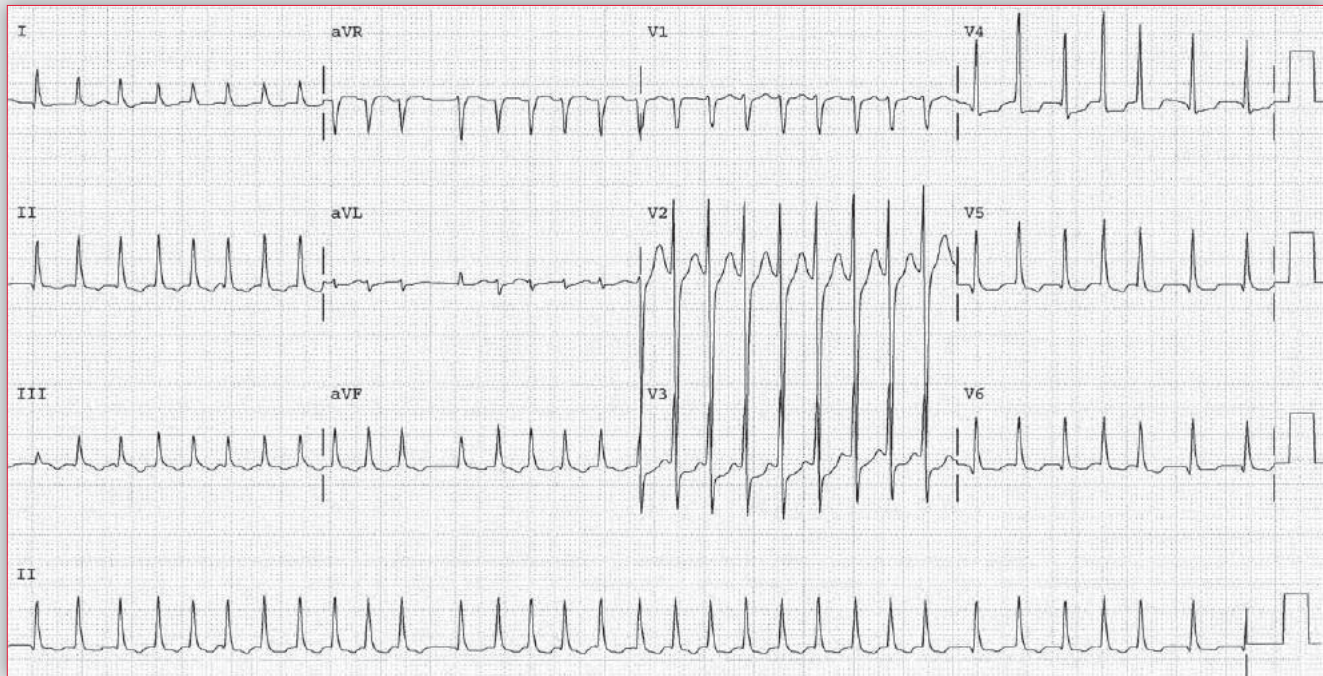
Notes

Practice Case 74

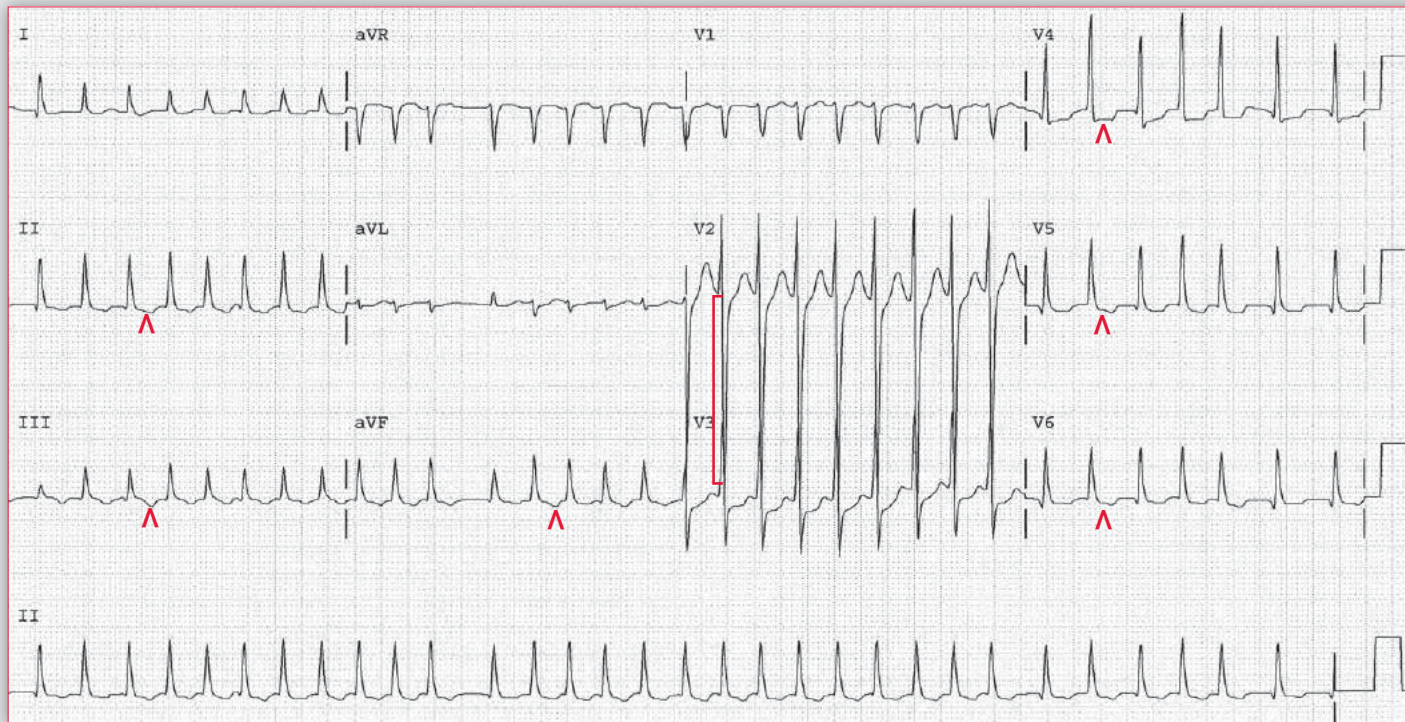
A 50-year-old man presents to the emergency department early on a Monday morning with complaints of palpitations and a rapid heart rate that began several hours before being seen. His history is unremarkable, although he does admit to heavy alcohol ingestion during the weekend as a result of personal problems. Physical examination is unremarkable except for the rapid heart rate. Blood pressure is 180/90 mm Hg. He appears to be slightly tremulous.

What does the ECG show?

What is the possible mechanism?



Podrid's Real-World ECGs



ECG 74 Analysis: Atrial fibrillation, left ventricular hypertrophy and nonspecific ST-T wave abnormalities

ECG 74 shows an irregularly irregular rhythm at a rate of 194 bpm. However, at rapid rates, the RR intervals may appear to be regular. In this situation, several RR intervals should be used for measurement and determining regularity, as small differences in the individual RR intervals become manifest. There are only three supraventricular arrhythmias that present with an irregularly irregular rhythm. These are sinus arrhythmia, in which there is only one P-wave morphology and PR interval; multifocal atrial rhythm (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm), in which there are > 3 different P-wave morphologies with no P-wave morphology being dominant and variable PR intervals; or atrial fibrillation, in which no organized P wave can be identified. In this ECG, there are no clear P waves seen. Therefore, this is atrial fibrillation with a very rapid ventricular response. The QRS complexes duration is normal (0.08 sec) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The S wave in lead V2 ([]) is 30 mm deep, and this fulfills one of the criteria for left ventricular hypertrophy (*ie*, R wave or S wave > 25 mm in any one precordial lead). There are ST-segment depressions (Δ) noted in leads II, III, aVF, and V4–V6, which are likely the result of left ventricular hypertrophy. The QT/QTc intervals are normal (220/400 msec).

Atrial fibrillation is an arrhythmia in which there is no organized atrial activity, but there are rapid fibrillatory waves within the right and left atria, occurring at rates of > 350 to 450 bpm and resulting from multiple small reentrant circuits. The ventricular rate is dependent upon AV nodal conduction. The normal AV node, when unstimulated, is capable of conducting impulses at rate of 120 bpm up to 170 bpm. A ventricular rate slower than 100 bpm is generally due to enhanced vagal tone, the use of an AV nodal blocking agent, or intrinsic AV nodal disease. A ventricular rate faster than this usually is due to enhancement of AV nodal conduction, which is the result of sympathetic activation or increased circulating catecholamines. A sympathetic state is, therefore, usually responsible for precipitating the arrhythmia. Etiologies include exercise, infection, hyperthyroidism, pulmonary embolism, or pulmonary edema. Another frequent cause is withdrawal from alcohol, which activates the sympathetic nervous system. A history of binge drinking over a weekend associated with the occurrence of atrial fibrillation on a Sunday night or Monday morning has been termed the “Holiday Heart Syndrome.” As sympathetic stimulation often plays a role, β-blockers are an effective therapy not only for blocking the AV node and slowing the ventricular rate, but also for potentially resulting in arrhythmia reversion. It is possible that underlying left ventricular hypertrophy and possible left atrial hypertrophy or a left atrial abnormality is another factor predisposing this patient to atrial fibrillation. ■

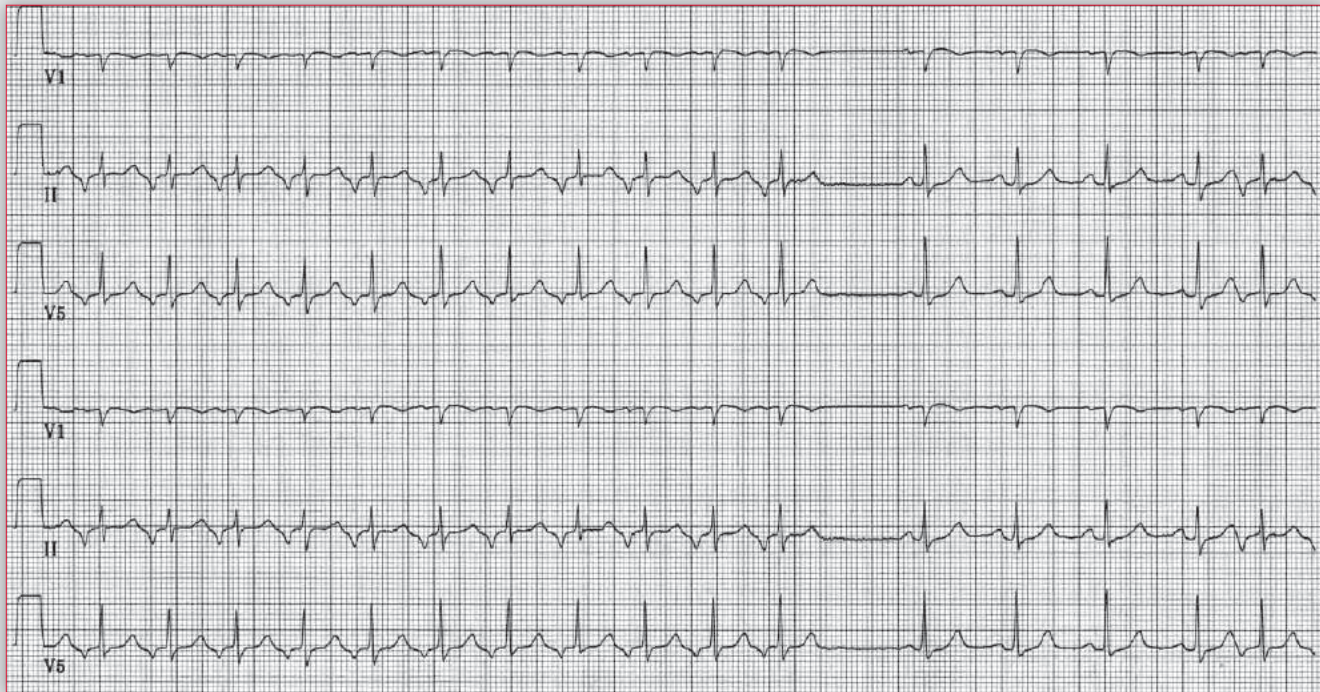
Notes

Practice Case 75

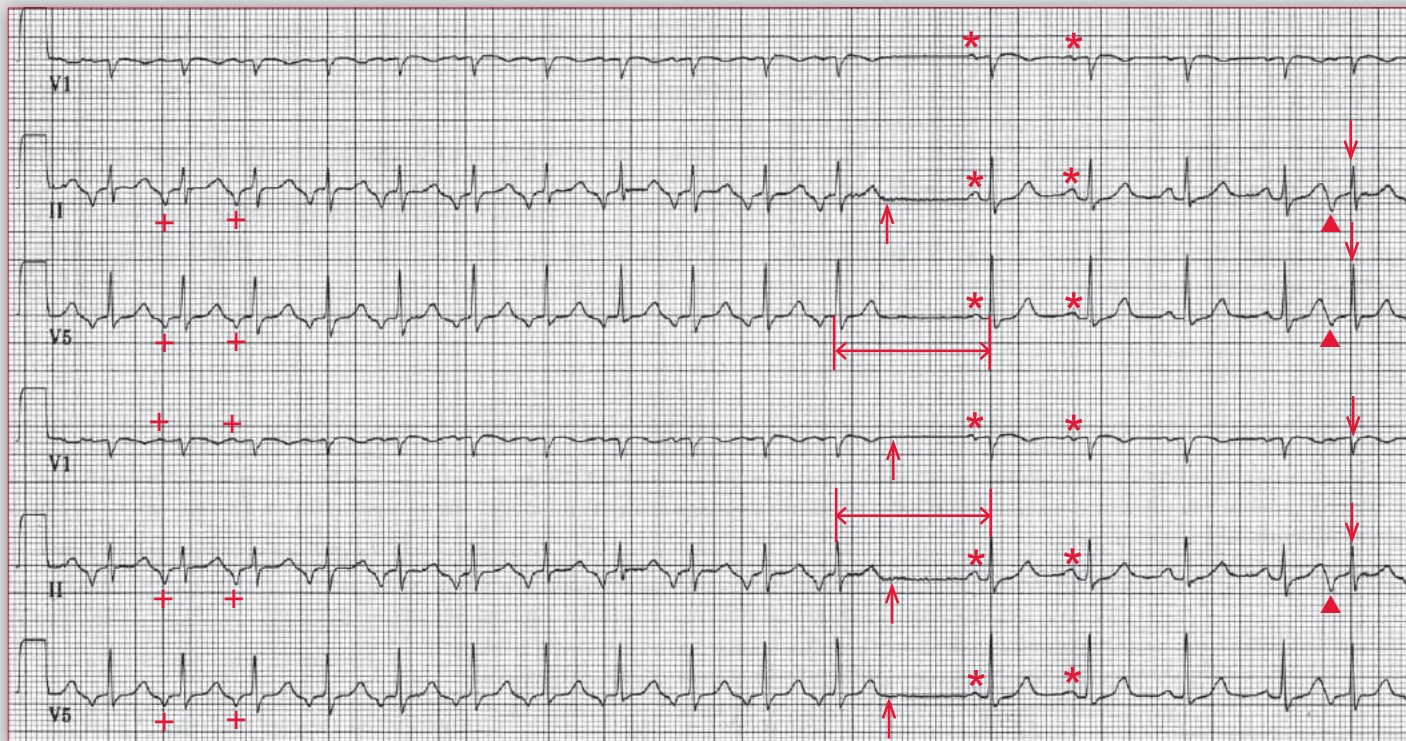
A 68-year-old man presents to his physician for a routine office visit. He has a history of hypertension, diabetes mellitus, and hyperlipidemia, but no cardiac disease. An ECG is obtained and the nurse notes an abnormality. Therefore, she obtained a rhythm strip.

What does the rhythm strip show?

Is any additional therapy necessary?



Podrid's Real-World ECGs



ECG 75 Analysis: Atrial tachycardia, normal sinus rhythm

ECG 75 shows the initial portion of the ECG rhythm strip (first 11 QRS complexes) featuring a regular rhythm at a rate of 120 bpm. The QRS complex duration is normal (0.08 sec) and there is likely a normal morphology, although only three leads are recorded. There is a P wave seen (+) before each QRS complex with a stable PR interval (0.18 sec). However, the P wave is negative in leads II and V5; therefore, it is not sinus in origin, but is an ectopic atrial focus. This is an ectopic atrial tachycardia. After the eleventh QRS complex, there is a pause (\leftrightarrow), after which there are similar QRS complexes at a slower rate (84 bpm). The QT/QTc intervals are normal (340/410 msec). There are P waves (*) before these QRS complexes with a stable PR interval (0.16 sec). The P waves have a different morphology compared to the P waves before the first 11 QRS complexes; they are positive in leads II and V5. Therefore, this is likely a sinus rhythm, which occurred after

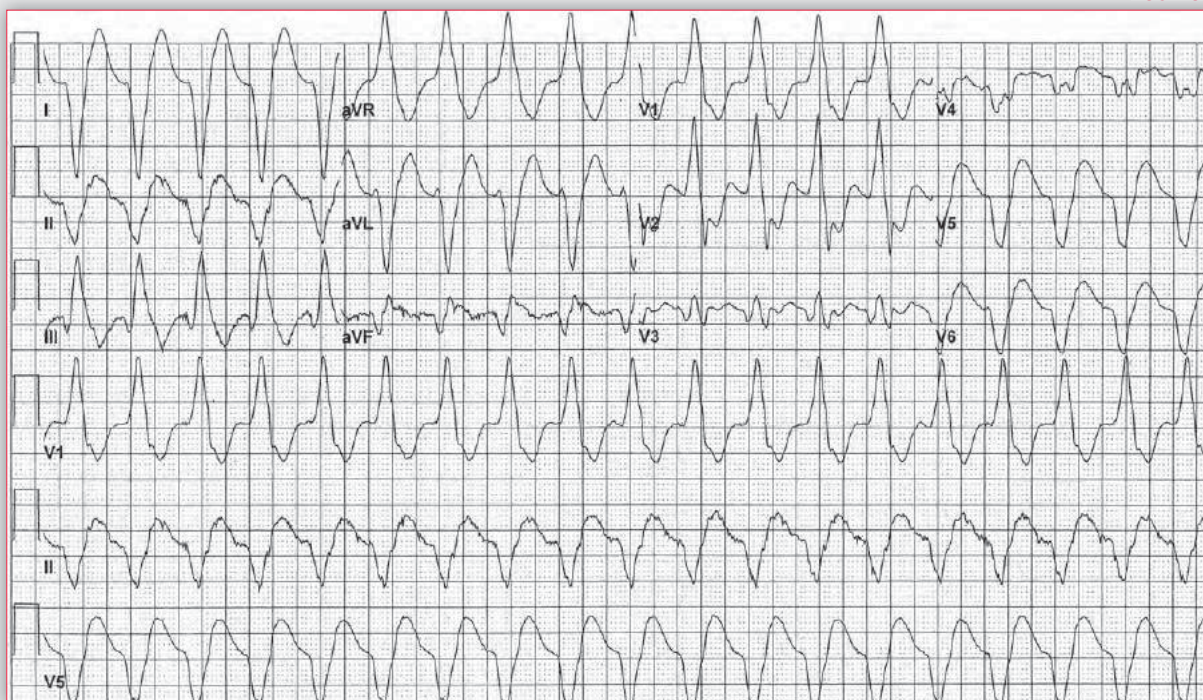
the abrupt termination of the atrial tachycardia. The tachycardia terminates with the absence of a P wave or atrial activity (\uparrow), which is the hallmark of atrial tachyarrhythmias. These arrhythmias terminate when the atrial focus abruptly stops and does not initiate any impulse; hence the absence of a P wave after the last QRS complex. The last QRS complex on the rhythm strip (\downarrow) is premature and has a negative P wave (\blacktriangle) before it. This is an ectopic atrial complex, and likely represents the initiation of recurrent atrial tachycardia.

An ectopic atrial tachycardia that is brief and self-terminating does not generally require any therapy. If the arrhythmia is of longer duration and associated with symptoms, chronic suppressive therapy may be necessary. The drugs of use are class IA, IC, and III antiarrhythmic agents. ■

Practice Case 76

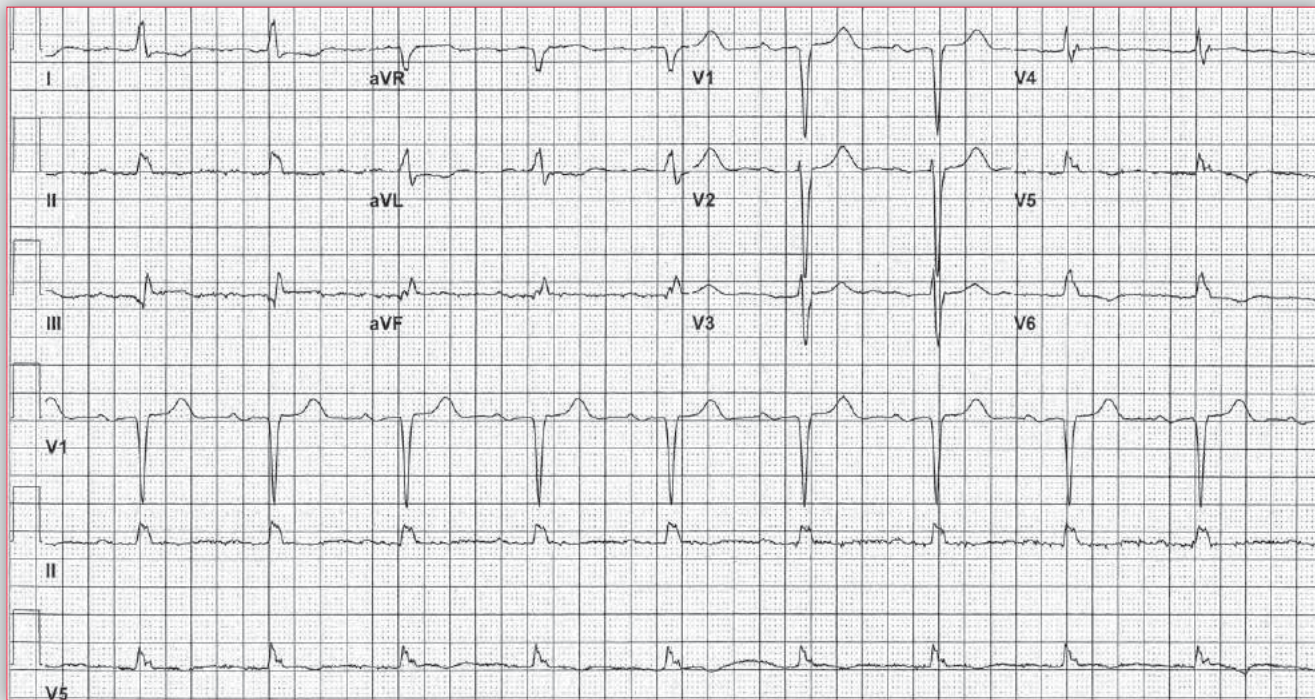
A 38-year-old man with a history of a familial cardiomyopathy develops an episode of pre-syncope while picking up his luggage at the airport. He is rushed to the nearest hospital, where the following ECG is obtained (ECG 76A). His blood pressure at the time is 80/60 mm Hg and he is complaining of feeling lightheaded and short of breath. As a result, he is urgently cardioverted. After cardioversion, another ECG is obtained (ECG 76B).

ECG 76A

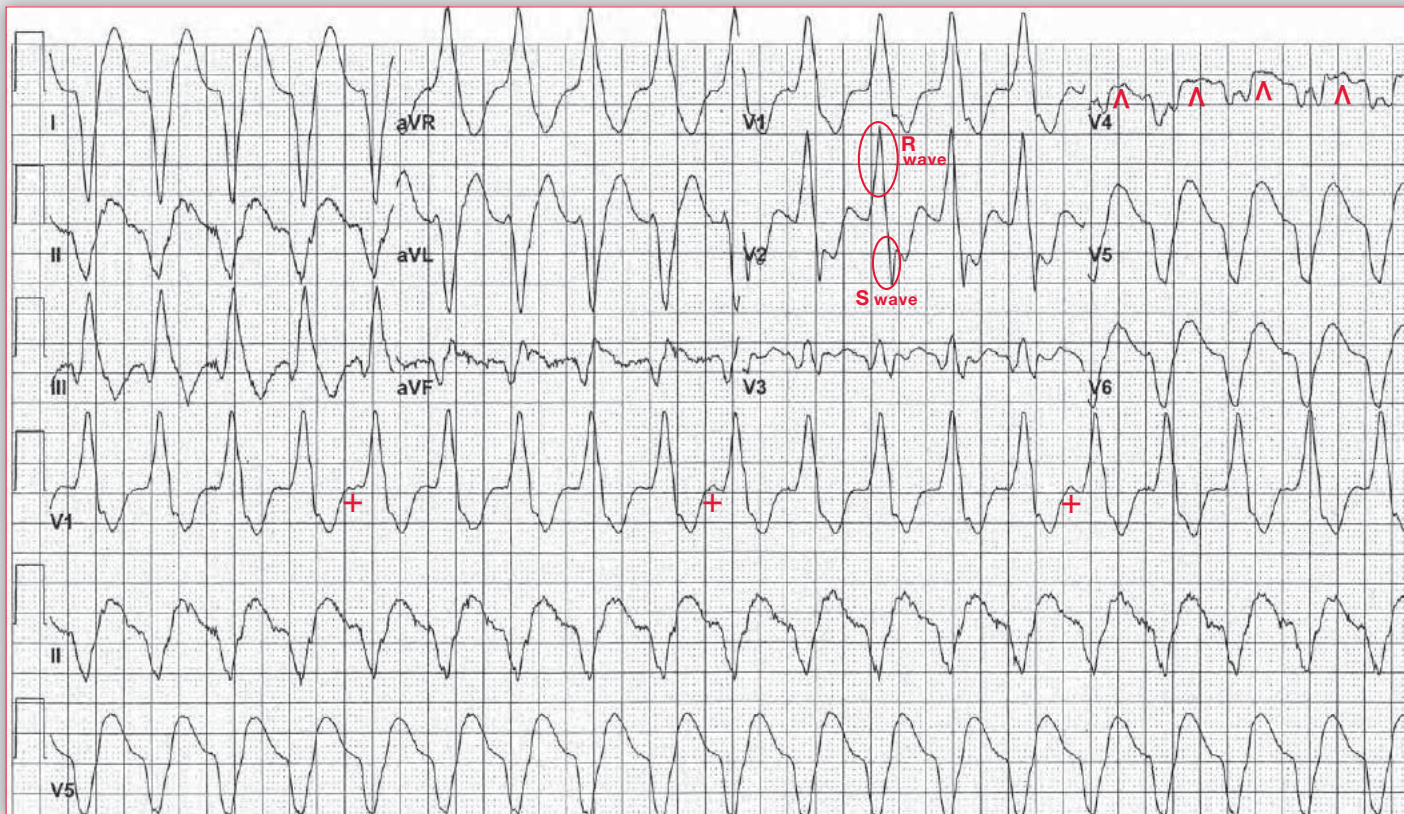


What is the etiology of the arrhythmia?

ECG 76B



Podrid's Real-World ECGs



ECG 76A Analysis: Wide complex tachycardia, ventricular tachycardia

ECG 76A shows a wide complex tachycardia (QRS duration = 0.18 sec) at a rate of 120 bpm. The QRS axis is indeterminate between -90° and $+/-180^\circ$ (negative QRS complex in leads I and aVF). The QRS morphology is abnormal and does not resemble either a right or left bundle branch block. There is no obvious atrial activity. However, careful inspection of the lead V1 rhythm strip shows subtle abnormalities between several of the QRS complexes that are suggestive of P waves; for example, between QRS complexes 4 and 5, 9 and 10, and 15 and 16 (+). Irregularities of the ST-T waves can also be seen in lead V4 (^). While these waveforms are not definitely P waves, they do represent irregularities or subtle changes of repolarization, which along with the indeterminate axis, are features of a ventricular tachycardia. In addition, there is a R/S complex in lead V2 and the R wave is much wider than the S wave ($R/S > 1$), which is also characteristic of a ventricular complex. An R wave that is wider than the S wave and > 100 msec in duration indicates that initial ventricular activation is abnormal and does not occur via the normal His-Purkinje system, but rather is due to conduction directly through the ventricular myocardium. This is, therefore, characteristic of a ventricular complex. When the R wave is narrower than the S wave or < 100 msec, the wide QRS complex is the result of aberration, *ie*, it is the terminal portion of ventricular activation that results from conduction directly through the ventricular myocardial. Since the QRS complexes are all the same, this is a monomorphic ventricular tachycardia.

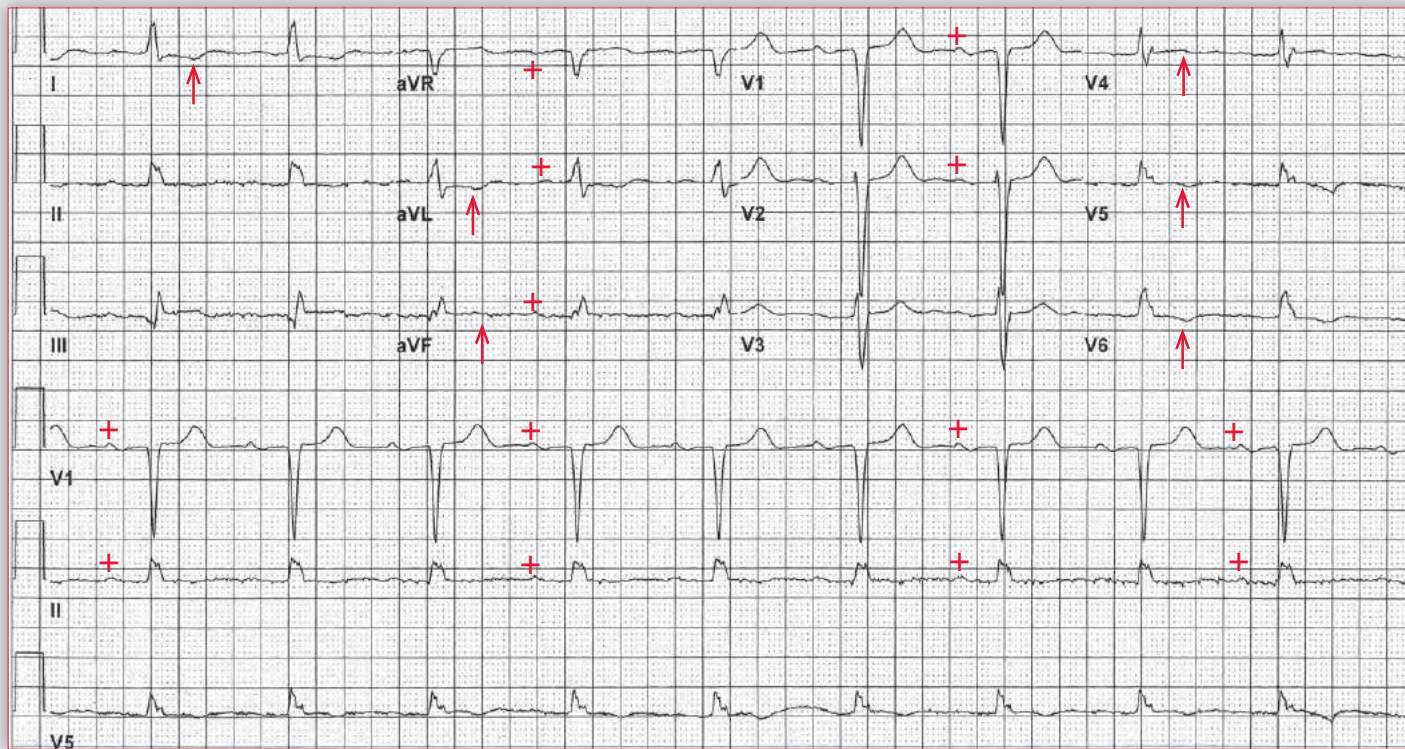
The most common mechanism for a sustained monomorphic ventricular tachycardia is reentry, the result of a circuit that develops

from damage to the myocardium and Purkinje fibers that can cause a disparity or heterogeneity of electrophysiologic properties (conduction and repolarization). This heterogeneity is a precondition for the development of reentry. In this patient, an underlying cardiomyopathy results in diffuse fibrotic changes within the myocardium that can cause this heterogeneity of electrophysiologic properties. Monomorphic ventricular tachycardia is not provoked by ischemia, but is scar- or fibrosis-related. It is often precipitated by electrolyte abnormalities, autonomic imbalance, an increase in circulating catecholamines, an increase in myocardial stretch among many other factors. These are conditions that can cause further changes in myocardial electrophysiologic properties, which can enhance the potential for reentry.

The most important ECG finding establishing ventricular tachycardia is the presence of a wide complex tachycardia with AV dissociation. This results from retrograde conduction of the ventricular impulse into the AV node but no retrograde activation of the atrial myocardium. As the AV node has been depolarized retrogradely at a rapid rate, the atrial impulse is unable to conduct antegradely through the AV node, resulting in dissociation between the atrial and ventricular impulses. Other important features are subtle differences in QRS morphology and ST-T waves, reflecting differences in the sequence pattern of myocardial activation and repolarization, as there is direct myocardial activation rather than activation through the His-Purkinje system, which is in a fixed sequence. With a supraventricular complex, there is uniformity of the QRS complexes and ST-T waves as the ventricular myocardial

continues

Podrid's Real-World ECGs



ECG 76B Analysis: Sinus bradycardia, first-degree AV block, intraventricular conduction delay, nonspecific ST-T wave abnormalities

activation sequence is always the same. Other important features include QRS complex morphology that does not resemble either a left or right bundle branch block and the presence of an indeterminate axis or positive concordance (tall R wave) across the precordium, which are only seen with any condition in which there is direct myocardial activation, *ie*, ventricular complex, Wolff-Parkinson-White, or ventricular pacing. A QRS complex > 0.16 second is generally not seen with an aberrated QRS complex, but is seen in a ventricular complex.

ECG 76B is from the same patient as ECG 76A. There is a regular rhythm at a rate of 58 bpm. A P wave (+) is present before each QRS complex with a stable and prolonged PR interval (0.28 sec). Therefore, this is a sinus bradycardia with a first-degree AV block or prolonged

AV conduction. The QRS complex duration is increased (0.12 sec), but there is no specific bundle branch block pattern; hence this is a non-specific intraventricular conduction delay. The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). There are also nonspecific ST-T wave abnormalities (\uparrow). The QT/QTc intervals are slightly prolonged (460/450 msec), but are normal when the prolonged QRS complex duration is considered (440/430 msec). When this ECG, which is the baseline ECG for this patient, is compared to ECG 76A, it can be seen that there have been marked changes in the QRS morphology, width and axis as a result of the ventricular tachycardia. ■

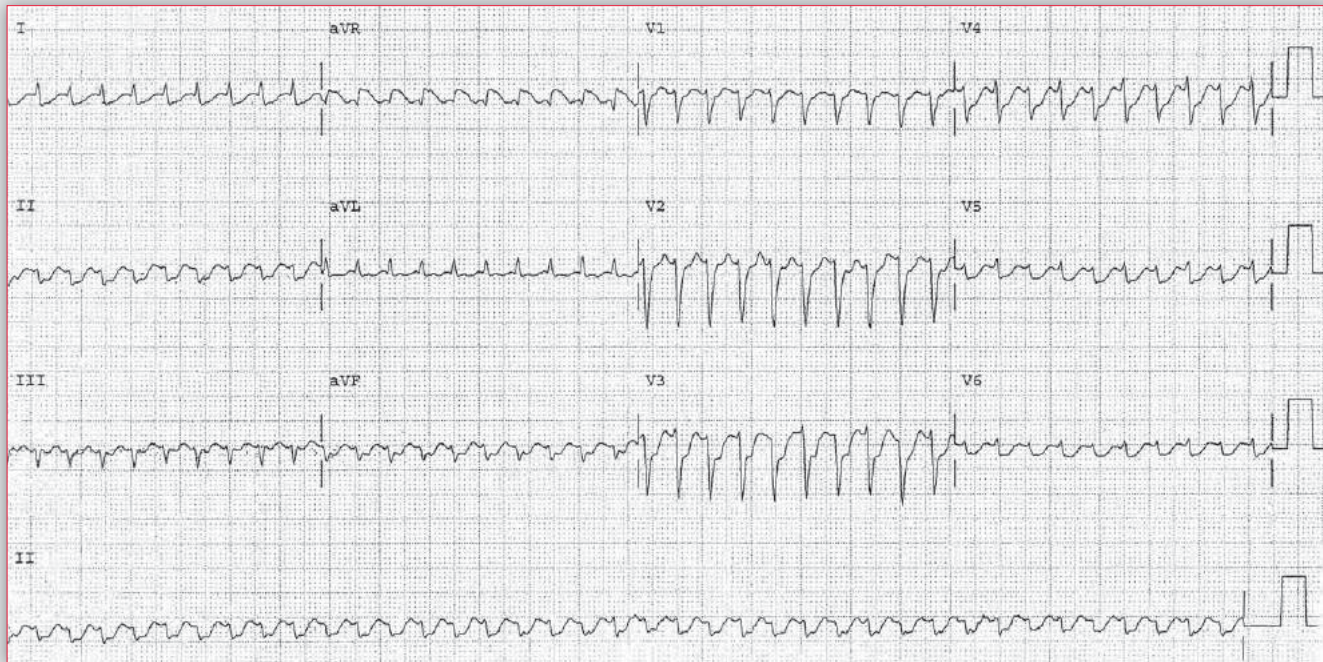
Notes

Practice Case 77

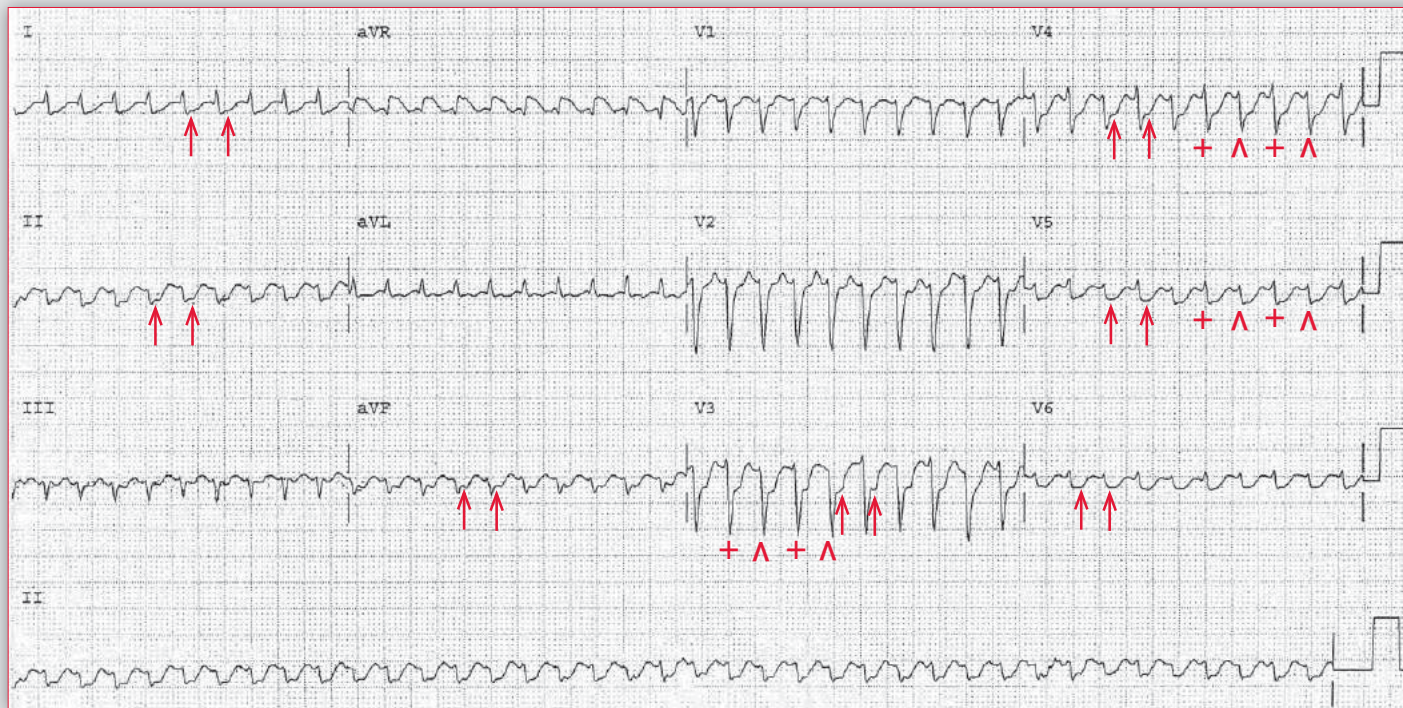
A 35-year-old man without any cardiac history notes that he has a persistently elevated heart rate after running in a 6-mile race. Although he does not complain of any major symptoms, he does note slight lightheadedness and shortness of breath that he initially attributed to running. He presents to an emergency department because of the elevated heart rate. An ECG is obtained.

What does the following ECG show?

What is the likely diagnosis?



Podrid's Real-World ECGs



ECG 77 Analysis: Narrow complex supraventricular tachycardia, atrial flutter with 1:1 AV conduction, left anterior fascicular block, low voltage in limb leads, electrical alternans, clockwise rotation

ECG 77 shows the rhythm is regular at a rate of 270 bpm. The QRS complex duration is normal (0.08 sec). The axis is extremely leftward between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF with an rS complex). This is a left anterior fascicular block. There is poor R-wave progression across the precordium, perhaps associated with a left anterior fascicular block. It may also be the result of clockwise rotation in the horizontal axis. This is established by imagining the heart as observed from under the diaphragm. With clockwise rotation, left ventricular forces develop late, becoming obvious in the left precordial leads. There is low voltage in the limb leads (< 5 mm in each lead). The QT/QTc intervals are normal (200/420 msec).

There are no obvious P waves seen before or after any QRS complex. However, this is a narrow complex, *ie*, supraventricular tachyarrhythmia. Atrial flutter is the only regular supraventricular tachyarrhythmia that presents with an atrial rate of ≥ 260 bpm (usually up to 320). Hence this is atrial flutter with 1:1 AV conduction.

In addition, there appears to be ST-segment depression (\uparrow) in leads V2–V6 as well as leads I, II, and aVF. Although subendocardial ischemia is potentially present at this heart rate, another possibility is that these ST-segment changes are actually the flutter waves.

Lastly, there are beat-to-beat changes in QRS complex amplitude, best seen in leads V3–V5 ($+, \wedge$). This is QRS (or electrical) alternans, and it is seen in several conditions, including any rapid and regular supraventricular tachyarrhythmia, severe dilated cardiomyopathy, decompensated heart failure, or an acute MI. In these situations, the electrical alternans is due to beat-to-beat changes in calcium fluxes into the myocardium. Alternans is also seen with cardiac tamponade, and in this situation, it is the result of swinging of the heart (pendulum effect) in a fluid-filled sac.

Atrial flutter with 1:1 AV conduction is uncommon as the normal AV node, in the absence of elevated catecholamines or sympathetic stimulation, is unable to conduct at rates above about 170 bpm. When there is sympathetic stimulation or an increase in circulating catecholamines, AV nodal conduction is enhanced, and the AV node is, therefore, capable of conducting impulses at a more rapid rate. In this situation, the patient developed atrial flutter during vigorous exertion.

Although the only supraventricular tachyarrhythmia that will occur at a rate > 260 bpm is atrial flutter, the diagnosis is often missed. However, it can be definitively established if AV nodal blockade is produced, exposing the atrial activity. This can be accomplished with an AV nodal blocking agent such as adenosine, a calcium-channel blocker, a β -blocker, or digoxin. ■

Notes

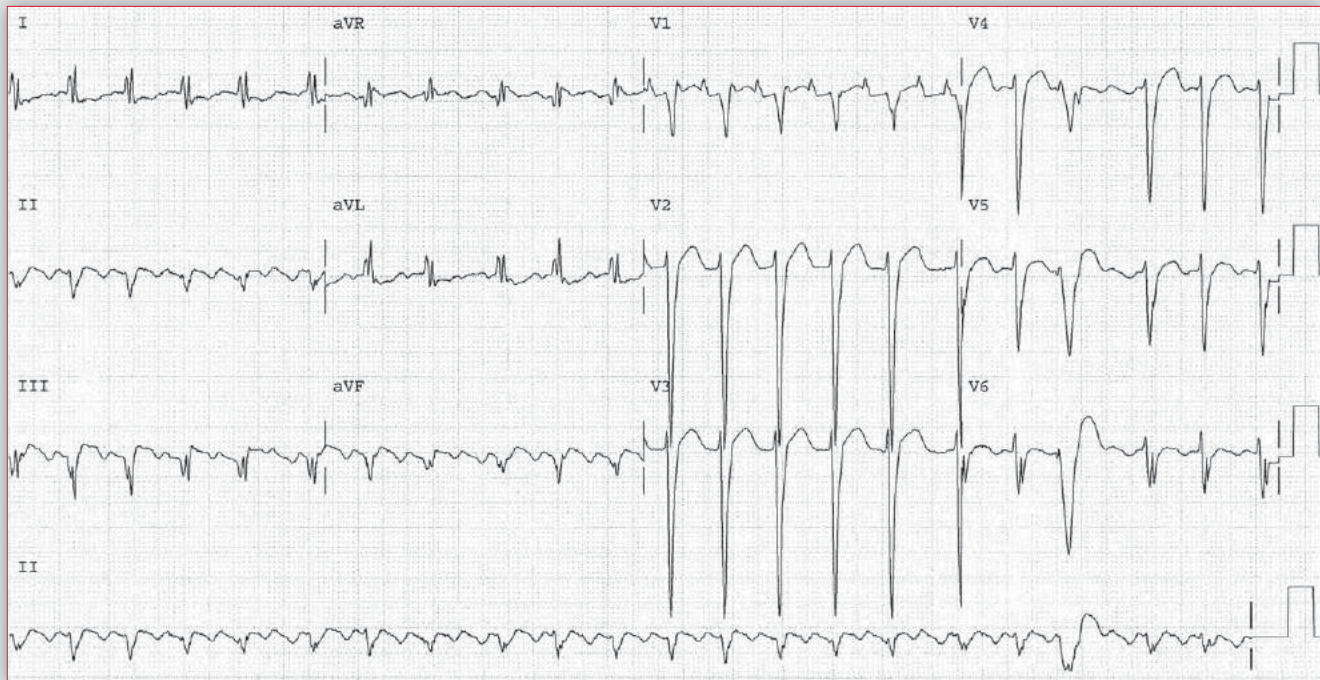
Practice Case 78

An 81-year-old man presents to his primary care physician with a racing heart. He has a history of paroxysmal atrial fibrillation for which he had been taking sotalol. He states that he has taken his pulse and noted that it has been rapid and regular, but at times, it is somewhat irregular.

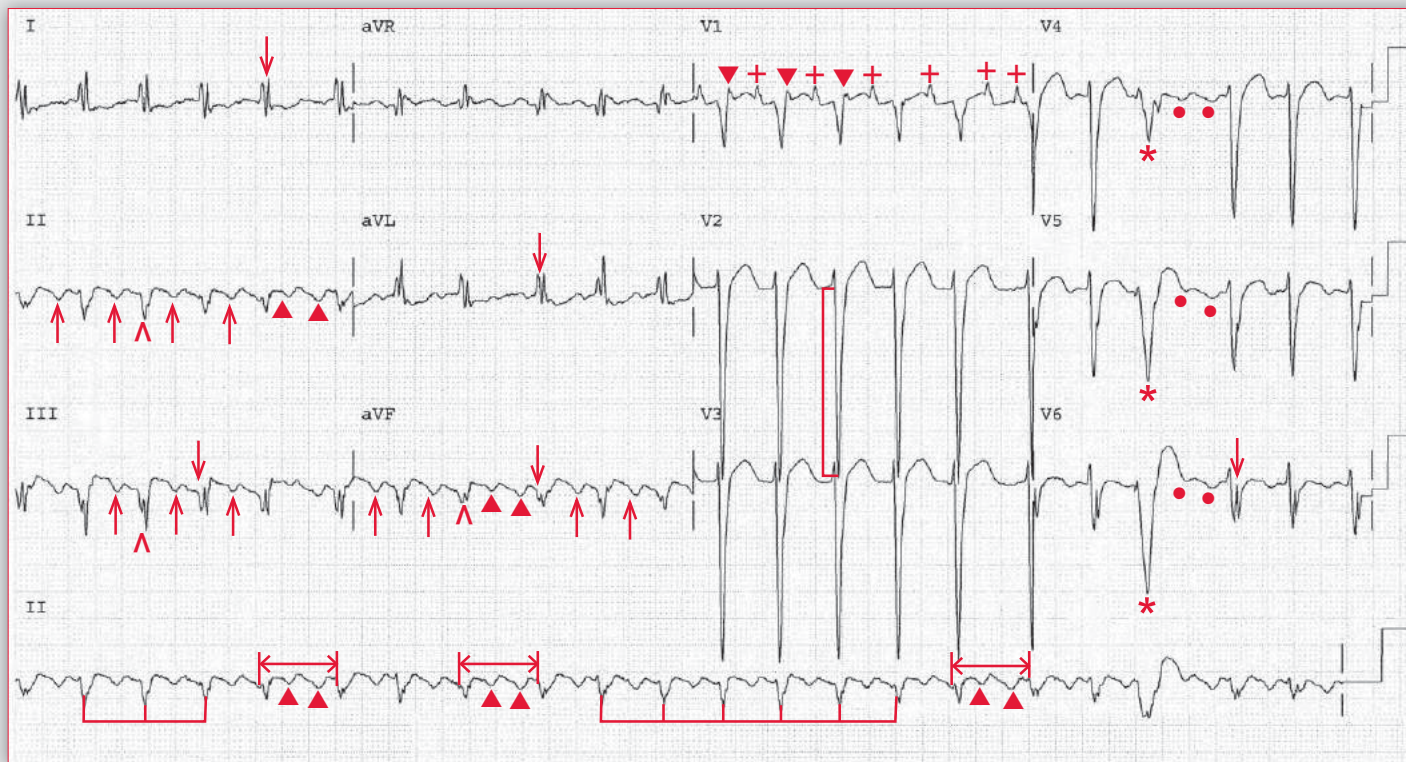
What does the ECG show?

What is the diagnosis?

Is there a relationship between his previous arrhythmia and what is noted on the present ECG?



Podrid's Real-World ECGs



ECG 78 shows a regularly irregular rhythm present as a result of longer RR intervals (\leftrightarrow), which all have the same duration, and shorter RR intervals (\sqcup), all of which are the same. The average rate is 132 bpm. The QRS complex duration is prolonged (0.12 sec), but there is no specific pattern present. Hence this is an intraventricular conduction delay. There is notching (\downarrow) of the QRS complex in leads I, III, aVL, aVF, and V6 that is consistent with the conduction delay. The axis is extremely negative between -30° and -90° (positive in lead I and negative in leads II and aVF). However, there are Q waves in leads II, III, and aVF (\wedge); hence this is an inferior wall myocardial infarction. In addition, there is poor R-wave progression in leads V1–V6, consistent with clockwise rotation. This is established by imagining the heart as if viewed from under the diaphragm. With clockwise rotation, left ventricular forces are delayed and are seen in later in the precordial leads. In addition, there increased QRS amplitude in lead V2 (S wave = 35 mm) (\sqcap); this is diagnostic of left ventricular hypertrophy (S wave or R wave in any precordial lead >25 mm), which is the reason for the intraventricular conduction delay and possibly the poor R-wave progression. The QT/QTc intervals are prolonged (300/425 msec) but are normal when the prolonged QRS complex duration is considered (300/425 msec). The nineteenth QRS complex (*) is premature, wide, and abnormal; this is a premature ventricular complex.

There is evidence of atrial activity that is best seen in lead V1 (\dagger). A second atrial waveform (\blacktriangledown), at a regular interval, is occasionally seen after the QRS complex. The atrial rate is regular at 280 bpm. The negative waveforms (\uparrow) in leads II, III, and aVF are atrial, and with the longer RR intervals, two atrial waveforms (\blacktriangle) can be seen at a rate of 280 bpm. Two atrial waveforms at a rate of 280 bpm can also be seen after the premature ventricular complex (\bullet). Therefore, the rhythm is atrial flutter with 2:1 and 3:1 AV block.

It is not uncommon for patients with atrial fibrillation to also have atrial flutter. Often atrial flutter reverts to atrial fibrillation. Reversion of atrial fibrillation to atrial flutter is less common. However, patients with atrial fibrillation treated with an antiarrhythmic drug often have recurrent arrhythmia in the form of atrial flutter as the antiarrhythmic drug stabilizes the atrium, preventing the multiple reentrant circuits; however, a single reentrant circuit may still exist, resulting in atrial flutter. Not uncommonly, the rate of the atrial flutter is slowed as a result of the antiarrhythmic drug, although the atrial flutter waves maintain their typical morphology, *ie*, they are undulating without an isoelectric baseline between each waveform. ■

Notes

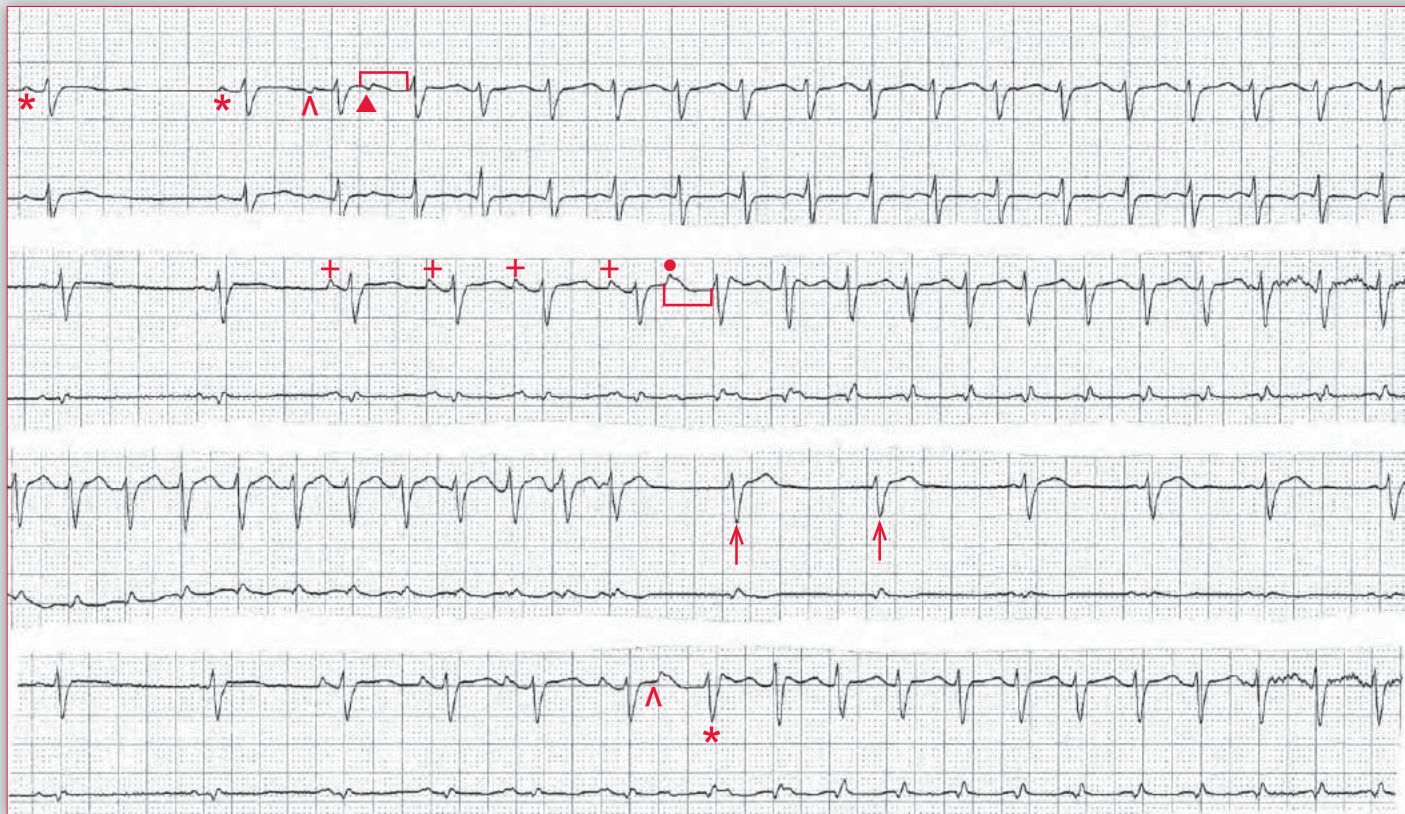
Practice Case 79

A 24-year-old woman with a history of paroxysmal palpitations is admitted to the hospital for an evaluation of abdominal cramps and diarrhea, felt to be the result of colitis. She complains of palpitations and as a result is placed on telemetry. During the night, she is noted to have episodes of tachycardia.

What arrhythmia is shown in the following set of rhythm strips?



Podrid's Real-World ECGs



ECG 79 Analysis: Sinus bradycardia, premature atrial complex, and narrow complex supraventricular tachycardia (no-RP tachycardia), atrioventricular nodal reentrant tachycardia

ECG 79 shows three different rhythm strips. On the first rhythm strip, the first two QRS complexes are sinus at a rate of 42 bpm. There is a P wave (*) before these QRS complexes with a PR interval of 0.18 second. The third QRS complex is premature. It has the same morphology and is preceded by a P wave (^) that has a different morphology, and there is a longer PR interval (0.24 sec). The fourth QRS complex, which is also premature, has the same morphology. There is a P wave before this complex (▲) that has the same morphology as the third P wave, but the PR interval is longer (0.32 sec) (□). Hence the third and fourth QRS complexes are premature atrial complexes. Following the fourth (and premature) QRS complex, there is a regular tachycardia at a rate of 140 bpm. The QRS complexes of the tachycardia have the same morphology as the first four QRS complexes. However, there are no P waves seen before or after any of these QRS complexes. Therefore, this is a narrow complex no-RP supraventricular tachycardia, and the most common etiology for this is an atrioventricular nodal reentrant tachycardia (AVNRT). The initiation of this tachycardia is typical for an AVNRT, *ie*, a premature atrial complex with a long PR interval. The mechanism for an AVNRT involves dual AV nodal pathways. The first pathway is a fast pathway that conducts rapidly but has a long refractory period and time for repolarization or recovery. The second pathway conducts the impulse slowly, but it has a short refractory period and repolarizes or recovers more quickly. These two pathways are linked proximally and distally in the AV node, forming a circuit. If there is a premature atrial impulse that enters the AV node before the fast pathway repolarizes or recovers, the impulse is conducted antegradely to the ventricles via the slow pathway, resulting in a long

PR interval. If the impulse reaches the distal end of the fast pathway at a time when the fast pathway has recovered, it will be conducted retrogradely back to the atria via this pathway at the same time it is conducted antegradely to the ventricles via the His-Purkinje system. If the retrograde impulse reaches the slow pathway when it has recovered, it will enter this pathway, again resulting in antegrade conduction to the ventricles. If this process continues, a reentrant tachycardia is established. As there is simultaneous activation of the atria and ventricles, no P wave is seen, *ie*, a no-RP tachycardia.

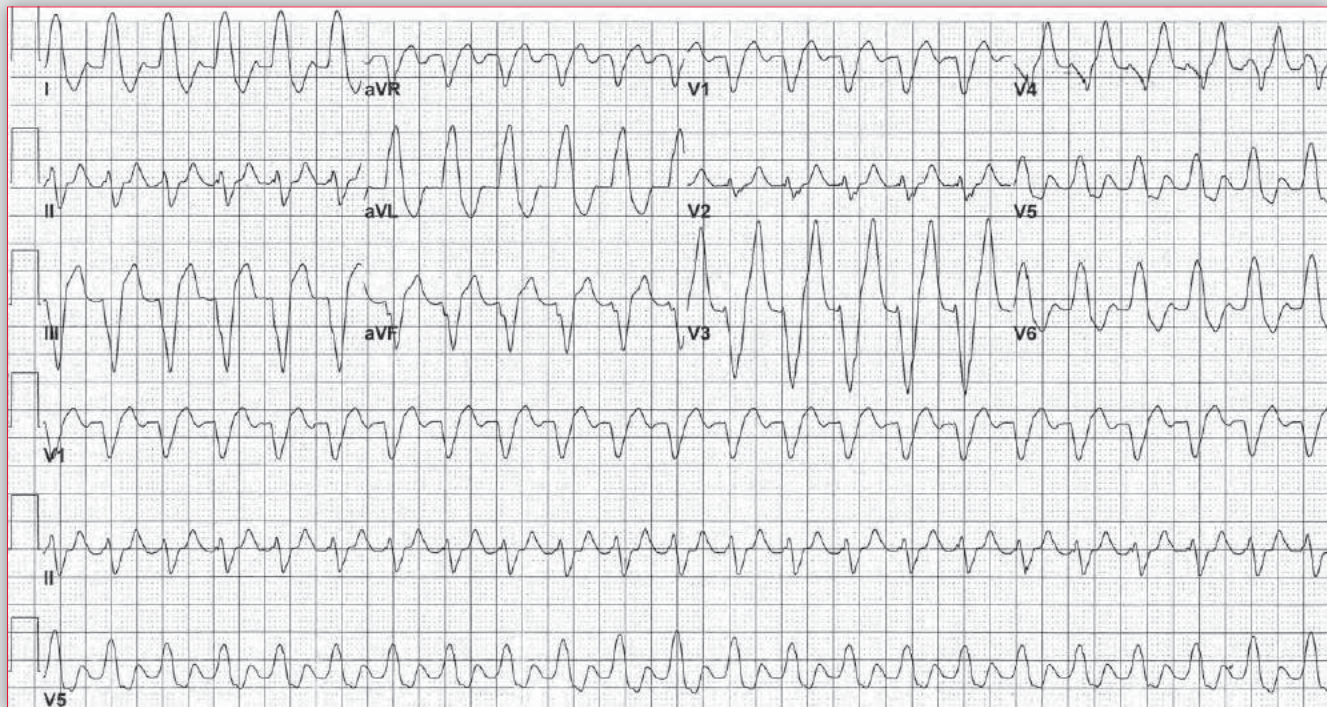
The second rhythm strip shows two sinus complexes at a rate of 56 bpm and a PR interval of 0.18 second. Complexes 3–6 have the same QRS morphology, but the preceding P waves (+) have a different morphology. Therefore, these are atrial complexes at a rate of 84 bpm. The seventh QRS complex is premature, occurring at a rate of 110 bpm. There is a P wave of a similar morphology seen before this QRS complex (●) but the PR interval has lengthened further (0.32 sec) (□). Following this complex is a regular tachycardia at a rate of 140 bpm. This is an AVNRT, similar to the tachycardia seen in the first rhythm strip. The third rhythm strip shows the abrupt termination of the AVNRT. The first two QRS complexes following the termination (†) do not have P wave before them; these are junctional escape complexes at a rate of 60 bpm. Following these, there is a sinus rhythm at a rate of 72 bpm.

The fourth strip again shows the onset of the AVNRT, which is a result of a premature atrial P (^) followed by a long PR interval and a premature QRS complex (*). ■

Practice Case 80

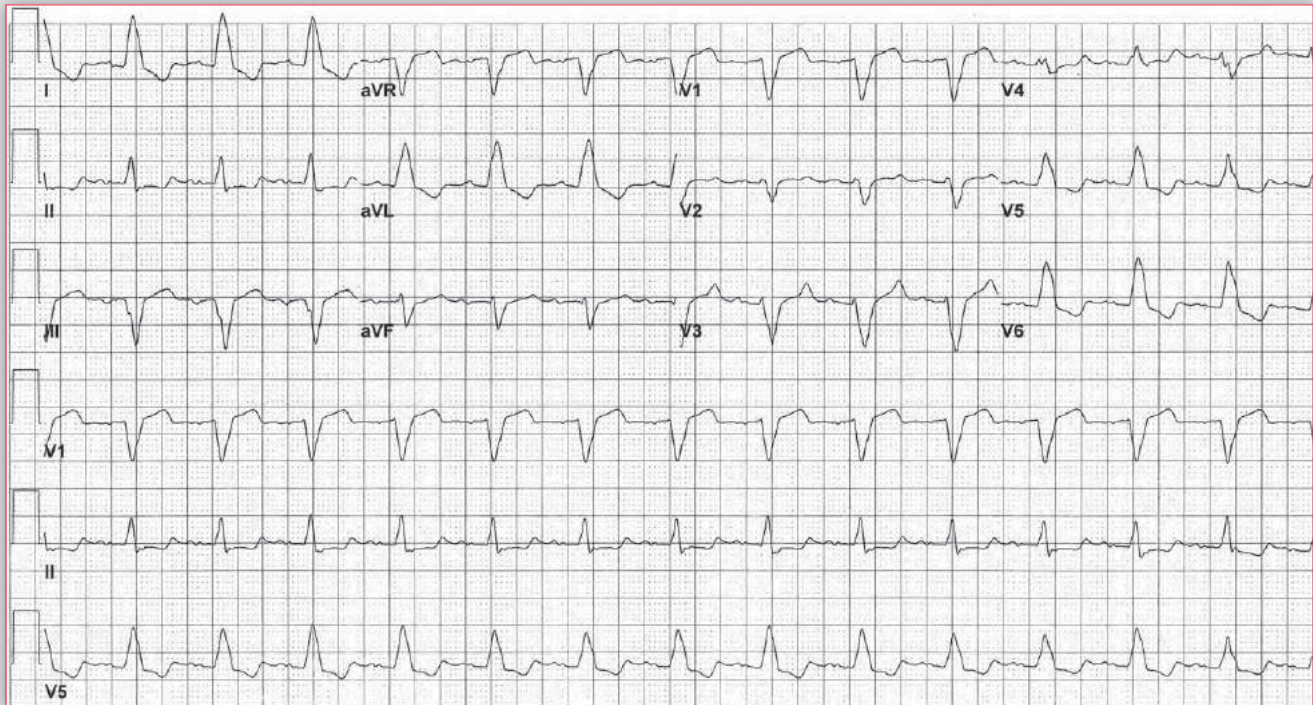
An 82-year-old man with no known cardiac disease presents with a two-day history of fever, cough, and shortness of breath. He denies chest pain or palpitations. Blood pressure is stable. A chest x-ray suggests the presence of pneumonia. An ECG is obtained (ECG 80A). This is compared to the patient's baseline ECG (ECG 80B).

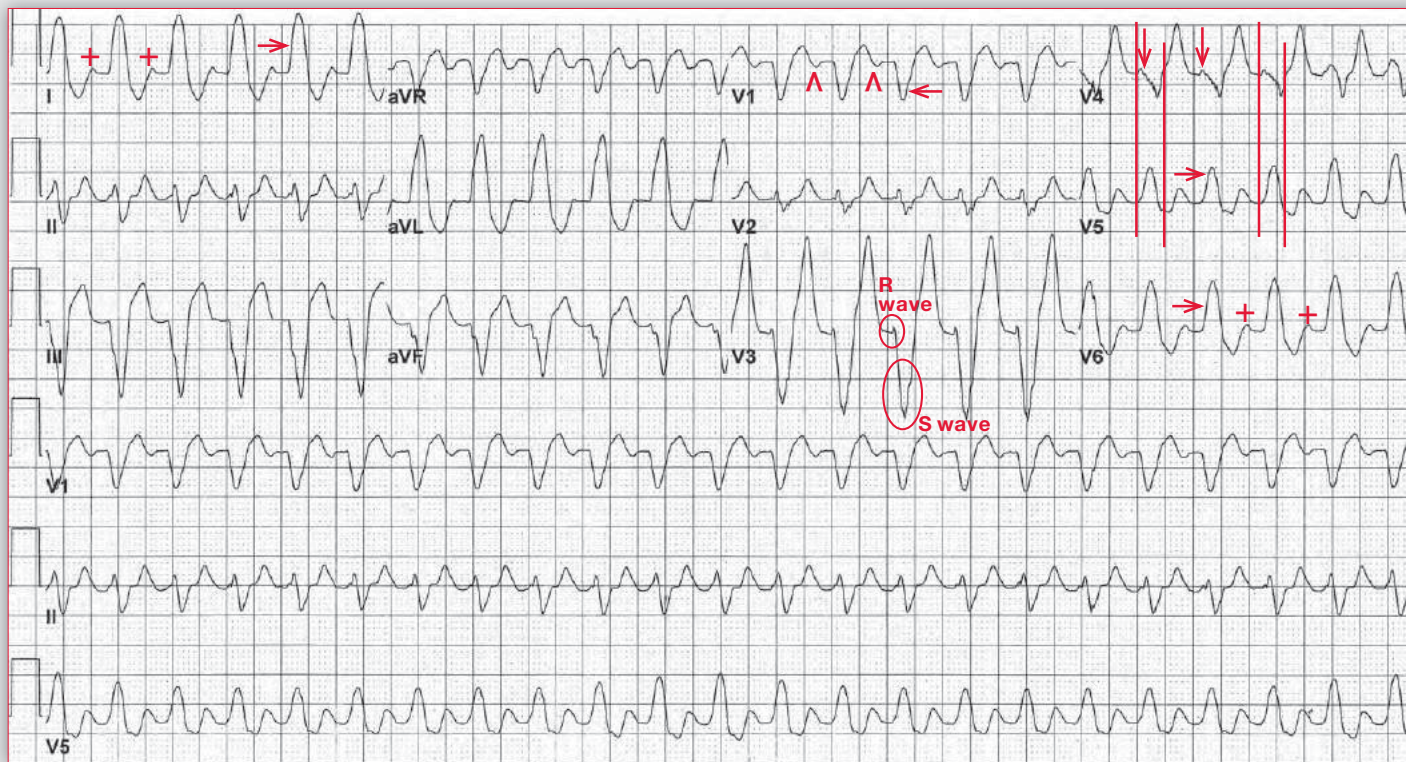
ECG 80A



What is the ECG diagnosis?

ECG 80B





ECG 80A Analysis: Wide complex tachycardia, sinus tachycardia, left bundle branch block

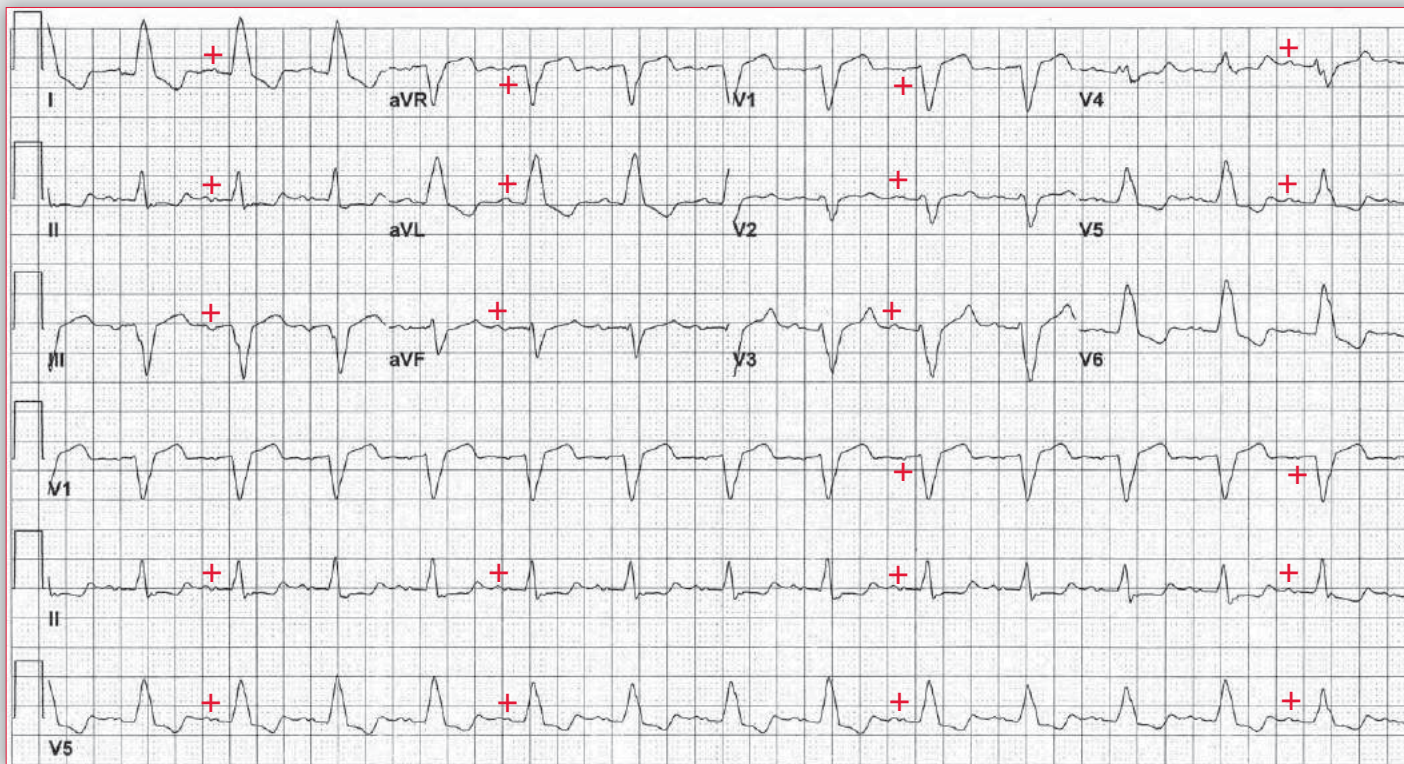
ECG 80A shows a regular rhythm at a rate of 148 bpm. There are no obvious P waves before or after any QRS complex. The QRS complex duration is increased (0.14 sec) and the morphology is a typical left bundle branch block (broad QS in V1 [←] and broad R wave in leads I and V5–V6 [→]). The axis is extremely leftward between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). The QT/QTc intervals are prolonged (320/500 msec) but is normal when the prolonged QRS complex duration is considered (280/430 msec). Although this is a typical left bundle branch block morphology, it cannot be definitively established if this is ventricular tachycardia or a supraventricular tachycardia with aberrancy. However, there is an R/S complex in lead V3, and the R wave is narrower than the S wave (and less than 100 msec). This suggests that this is a supra-ventricular rhythm with aberration. With aberration (either right or left bundle branch block) the initial activation of the ventricle is via the normal His-Purkinje system; hence the initial forces (*ie*, R wave) are

normal in duration, while the terminal forces (S wave) are prolonged and abnormal, as the terminal activation is not via the normal Purkinje system but is directly through the ventricular myocardium. With a ventricular complex, all of ventricular activation is abnormal, *ie*, there is direct myocardial activation. Hence the R wave is wider than the S wave and > 100 msec in duration.

While P waves are not obvious, there is a positive deflection at the end of the T wave in lead I and V6 (+) as well as a negative deflection at the end of the T wave in lead V1 (^). It is possible that these deflections may be P waves, although they may also represent the terminal portion of the T wave. If a P wave, then the PR interval is 0.18 second. The positive waveform seen at the beginning of the QRS complex in lead V4 (↓) is actually part of the QRS complex and not a P wave, as established by comparing the QRS width in lead V5 with this QRS complex (||).

continues

Podrid's Real-World ECGs



ECG 80B Analysis: Normal sinus rhythm, left atrial hypertrophy, left bundle branch block

ECG 80B is from the same patient as ECG 80A. There is a regular rhythm at a rate of 84 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The P waves are broad and notched in leads II and V3–V6, suggesting left atrial hypertrophy or a left atrial conduction delay. The QRS complex duration (0.14 sec) and morphology are identical to what was seen in ECG 80A, and there is a left bundle branch block present. The QT/QTc intervals are prolonged (400/470 msec), but are normal when the prolonged QRS complex duration is considered (360/420 msec). Hence the arrhythmia in ECG 80A is supraventricular with a left bundle branch block that is preexistent, being present during normal sinus rhythm.

As indicated, it is not clear if there are P waves seen in ECG 80A. However, there were positive waveforms seen that suggested P waves. By comparing the T-wave morphologies in ECGs 80A and 80B, it can be seen that the positive waveform at the end of the T wave in ECG 80A

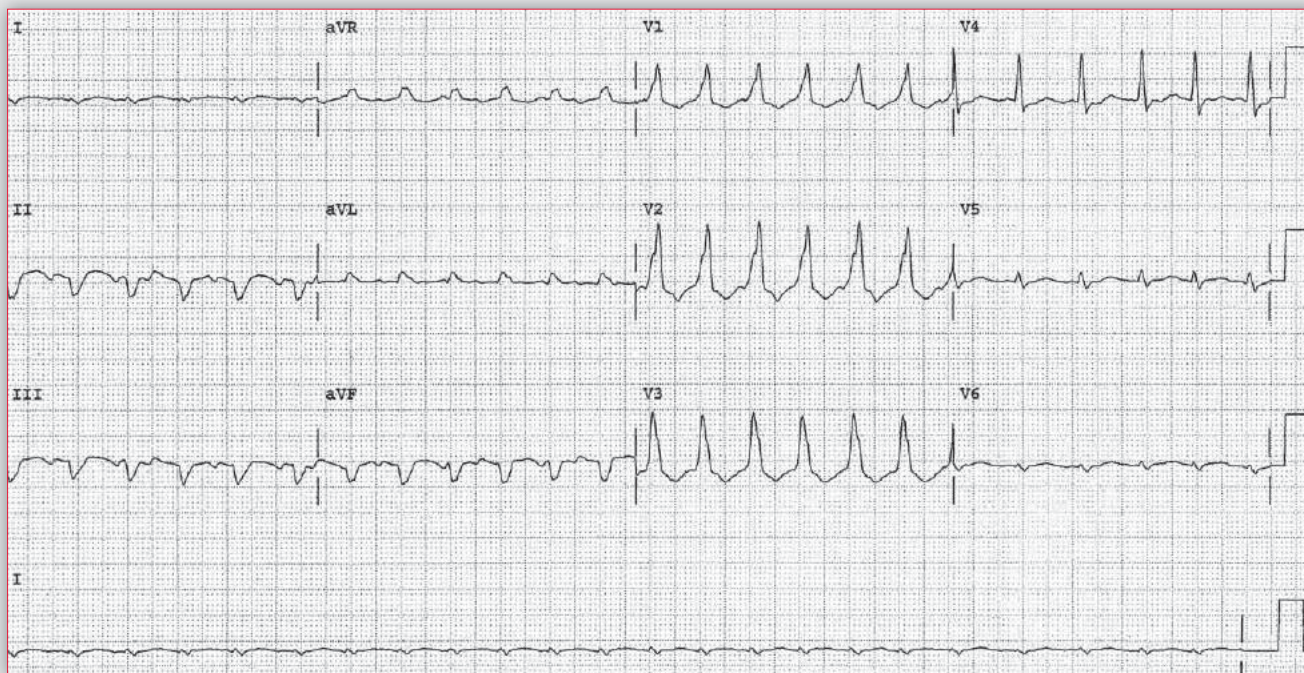
is not seen in ECG 80B, further supporting the fact that this is indeed the P wave superimposed at the end of the T wave as a result of the tachycardia. Indeed, if these are P waves, the PR interval in ECG 80A is slightly shorter than the PR interval seen in ECG 80B (*ie*, 0.18 vs. 0.20 sec). This is consistent with the change in the PR interval at different sinus rates (*ie*, 148 vs. 84 bpm), since PR intervals usually shorten with sinus tachycardia due to sympathetically mediated enhanced conduction through the AV node. Therefore, the PR intervals are similar, and thus the rhythm in ECG 80A is consistent with a sinus tachycardia. The left bundle branch block is not rate-related, but is preexistent.

Sinus tachycardia is generally related to activation of the sympathetic nervous system and an increase in circulating catecholamines; hence it is a physiologic rhythm in response to a condition that is associated with sympathetic activation. In this patient, it is in response to a pneumonia. ■

Practice Case 81

A 74-year-old woman with known CAD and Class IV chronic renal failure presents to the ED with palpitations. An ECG is obtained (ECG 81A). A baseline ECG is obtained after the arrhythmia is treated (ECG 81B).

ECG 81A



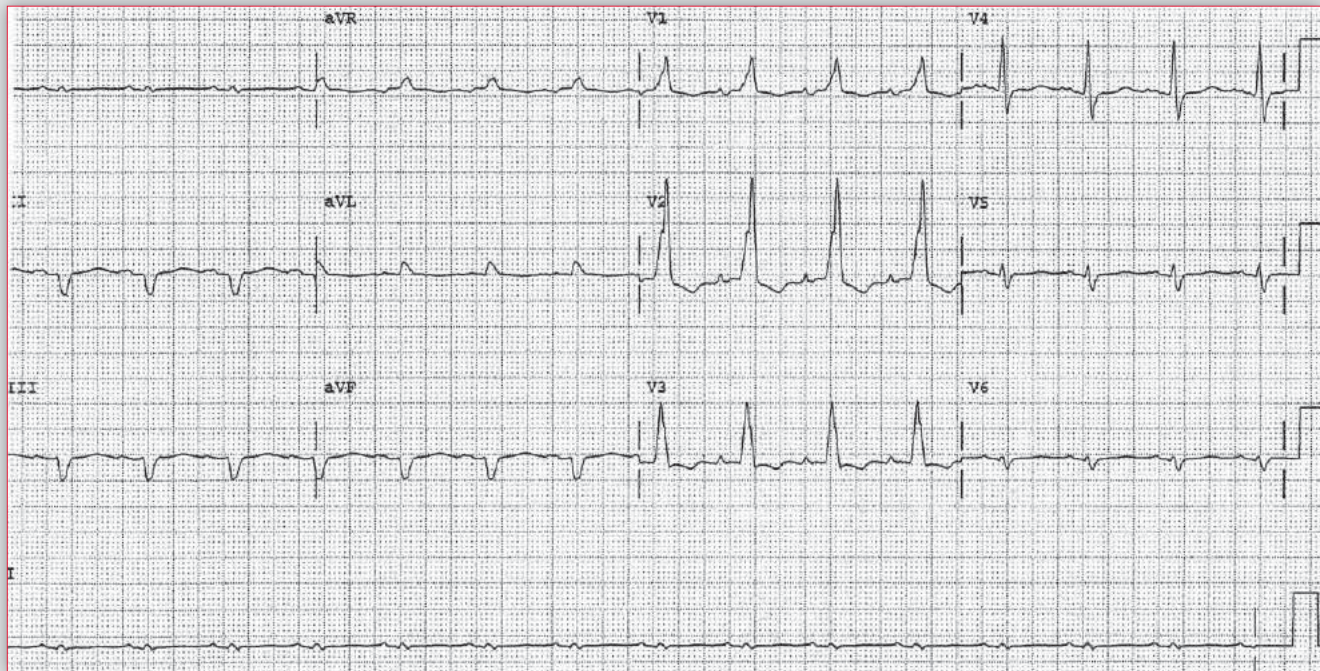
Practice Case 81

What is the rhythm disturbance in the initial ECG?

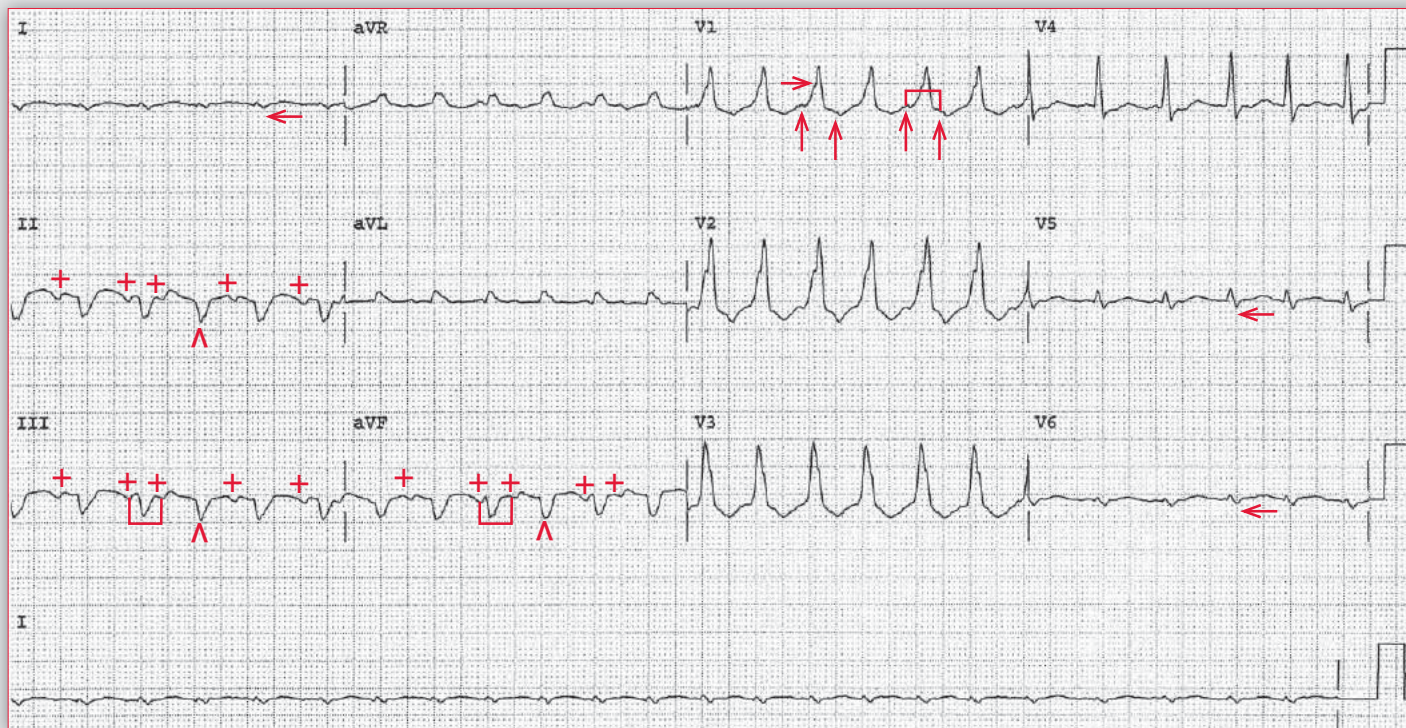
Which of the following agents is most appropriate for chronic therapy of this arrhythmia?

A. Amiodarone B. Flecainide C. Mexiletine D. Propafenone E. Sotalol

ECG 81B



Podrid's Real-World ECGs



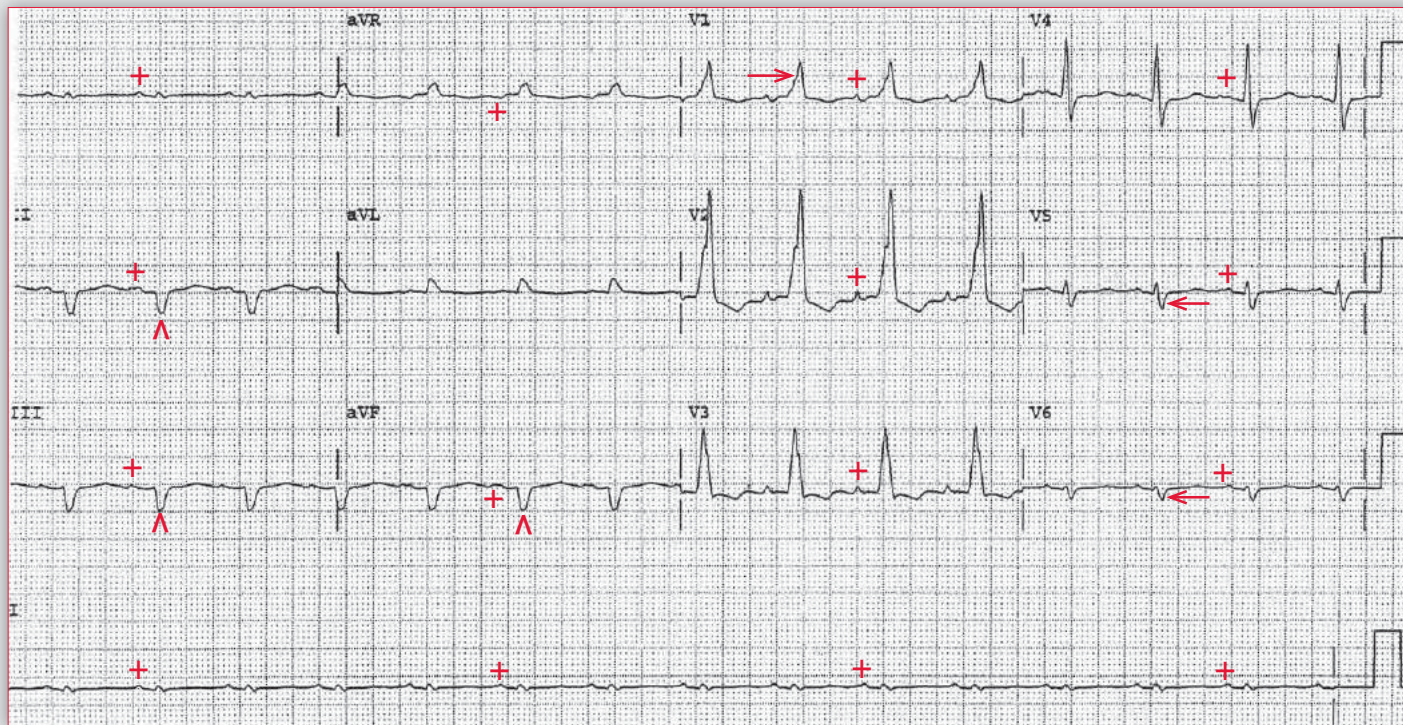
ECG 81A Analysis: Atrial flutter with 2:1 AV conduction, right bundle branch block, old inferior MI

ECG 81A shows a regular rhythm at a rate of 150 bpm. The QRS complex duration is increased (0.16 sec) and there is a right bundle branch block morphology with a broad R wave in lead V1 (→) and broad S wave in leads I and V5–V6 (←) (although the QRS complex amplitude in this lead is very small). The axis is extremely leftward between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF). However, the leftward axis is due to Q waves in leads II, III, and aVF (^), diagnostic of an old inferior wall myocardial infarction. The QT/QTc intervals are prolonged (320/510 msec), but are normal when the prolonged QRS complex duration is considered (260/400 msec).

Although there are no obvious P waves seen, there are negative waveforms in leads II, III, and aVF (+) that are suggestive of P waves that are superimposed on the ST segments and T waves. A negative waveform can be seen before and after the second complex in lead III and the third QRS complex in aVF. Using this as a PP interval (⊥), it can be seen that these waveforms occur at regular intervals at a rate of 300 bpm. These waveforms, at the same regular rate (⊐), can also be seen in lead V1, for example before and after the third and fifth QRS complexes in this lead (†). As the atrial rhythm is regular at a rate of 300 bpm, the underlying rhythm is atrial flutter and with the ventricular rate of 150 bpm, there is 2:1 AV conduction.

continues

Podrid's Real-World ECGs



ECG 81B Analysis: Sinus rhythm, right bundle branch block, old inferior MI

ECG 81B is from the same patient as ECG 81A. The rhythm is regular at a rate of 90 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, V4–V6. Hence this is a sinus rhythm. The QRS complex duration (0.16 sec) and morphology is the same as seen in ECG 81A, *ie*, there is a right bundle branch block with a broad R wave in lead V1 (→) and S wave in leads V5–V6 (←) and a chronic inferior wall myocardial infarction, with Q waves in leads II, III, and aVF (^). Hence the right bundle branch block during the atrial flutter is not rate-related, but rather it is preexistent, being present during sinus rhythm and a slower rate. The QT/QTc intervals are prolonged (380/470 msec) and normal when the prolonged QRS complex duration is considered (320/390 msec).

The best antiarrhythmic agent in this patient for long-term chronic therapy to prevent recurrent atrial flutter would be amiodarone, a class III antiarrhythmic drug. The class IC antiarrhythmic agents, *ie*, flecainide or propafenone, are not appropriate agents in patients with structural heart disease, as is the case with this patient who has had a previous myocardial infarction. Sotalol, another class III antiarrhythmic agent, should not be used in patients with significant renal failure, as this drug is cleared unchanged by the kidney. Mexiletine is a class IB agent that is not effective for atrial arrhythmias and is indicated only for ventricular arrhythmias. ■

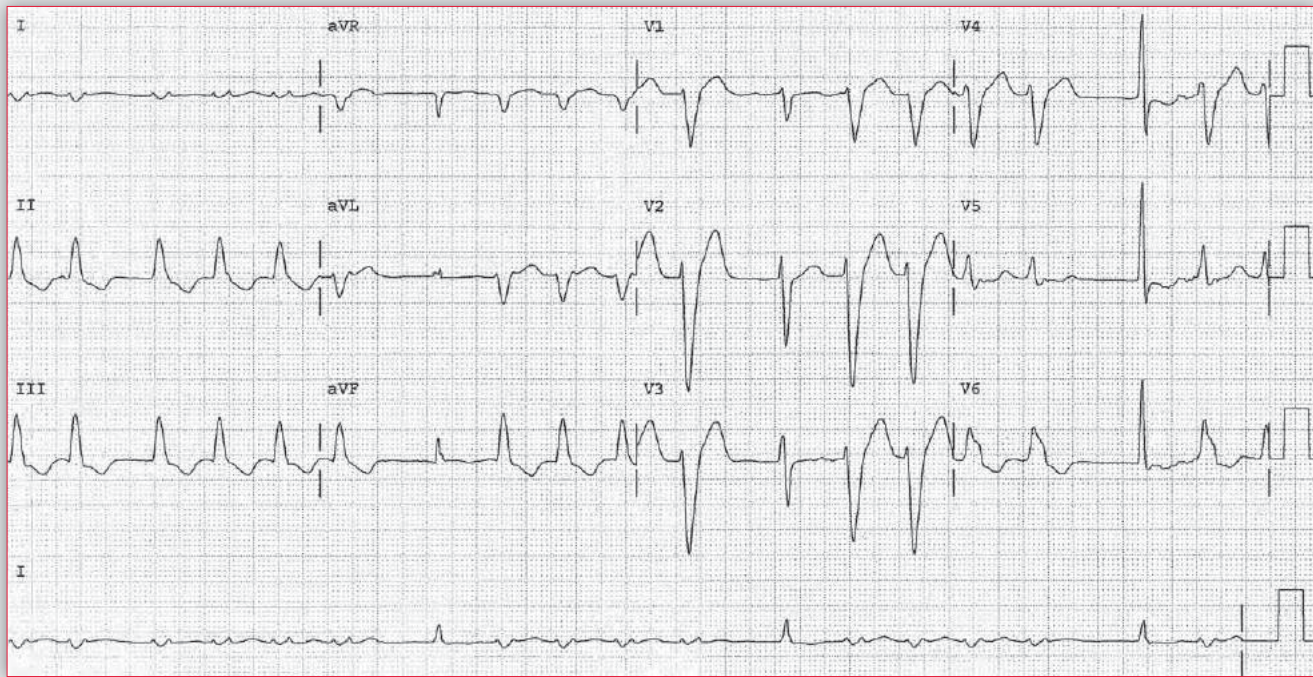
Notes

Practice Case 82

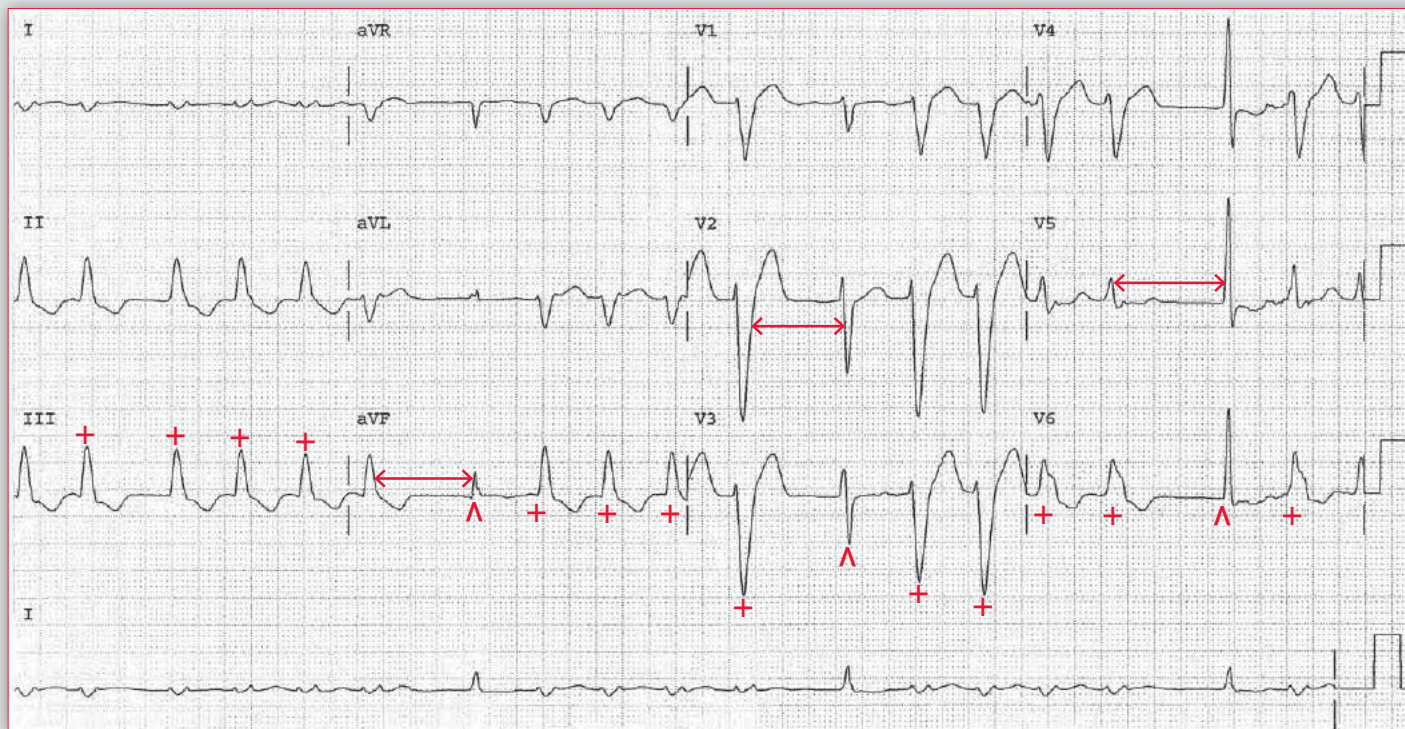
A 63-year-old man with obstructive hypertrophic cardiomyopathy who underwent recent septal myomectomy presents with palpitations. He is admitted to the hospital for observation and on the following day he again complains of palpitations. The following ECG is obtained.

What is the arrhythmia?

What is the mechanism for the two different QRS morphologies?



Podrid's Real-World ECGs



ECG 82 Analysis: Atrial fibrillation, rate-related left bundle branch block

ECG 82 shows the rhythm is irregularly irregular at a rate of 110 bpm and there are no obvious P waves seen before or after any QRS complex. There are only three supraventricular arrhythmias that are irregularly irregular. These are sinus arrhythmia, in which there is one P-wave morphology and one PR interval; multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm), or multifocal atrial tachycardia (rate > 100 bpm), in which there are > 3 different P-wave morphologies with no P-wave morphology being dominant and variable PR intervals; and atrial fibrillation, in which there is no organized atrial activity or P wave. Atrial flutter and atrial tachycardia may be irregular, but the irregularity is based on the degree of AV block; hence when irregular, these arrhythmias are said to be regularly irregular. Therefore, this is atrial fibrillation. There are narrow and wide QRS complexes. The wide complex (+) duration is 0.16 second and the morphology is that of a left bundle branch block. The narrow QRS complexes (^) have a normal duration (0.08 sec) and morphology. The RR interval preceding each narrow QRS complex is long (slow rate) (↔), while the QRS complexes with a left bundle branch block always occur with a shorter

RR interval (faster rate). Hence this is atrial fibrillation at a rate-related left bundle branch block. The QT/QTc intervals are slightly prolonged (340/460 msec).

A rate-related bundle branch block, also termed a functional bundle branch block, results from an underlying conduction abnormality of the bundle such that it is unable to conduct above a certain heart rate, leading to a failure of the bundle to conduct the impulse. The rate at which a bundle branch block develops may vary from one day to the next. In addition, the rate at which the block develops may be different than the rate at which the block resolves and normal conduction is seen. A rate-related or functional bundle branch block may present with either a right or a left bundle branch block pattern and may even present only with a left anterior or left posterior fascicular block or even a nonspecific intraventricular conduction delay. A rate-related bundle branch block may predict the occurrence of a permanent bundle branch block in the future. ■

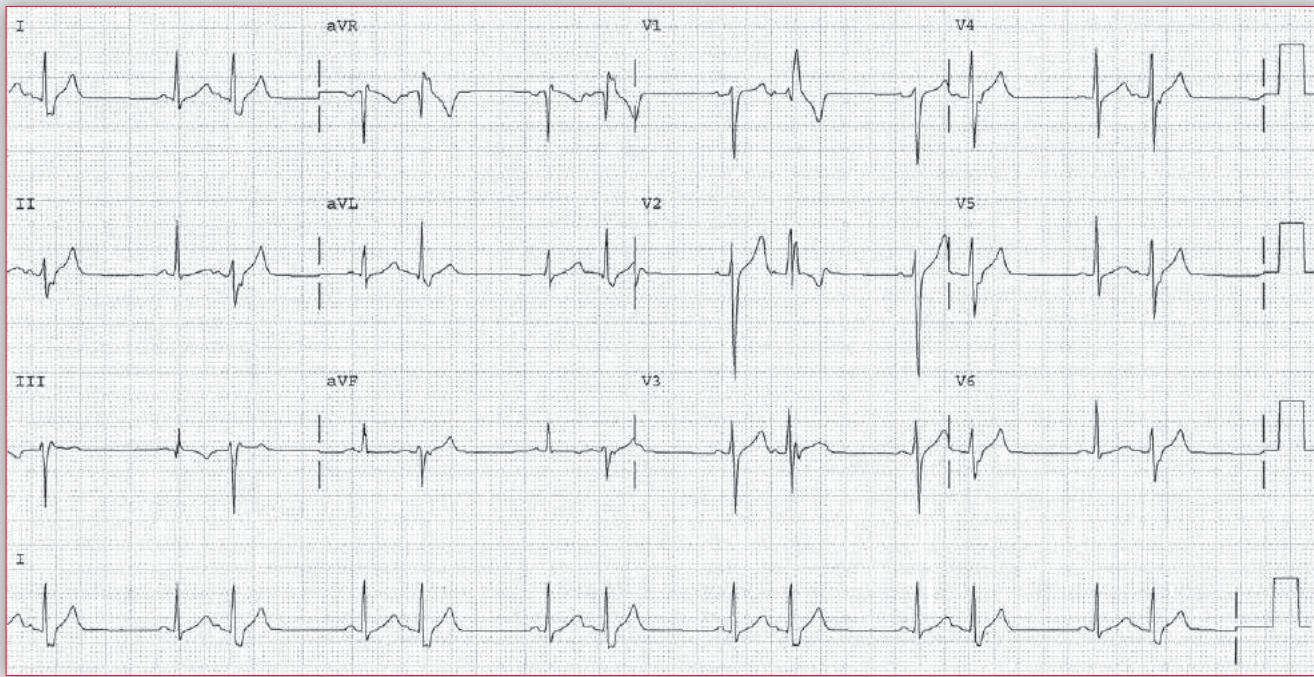
Notes

Practice Case 83

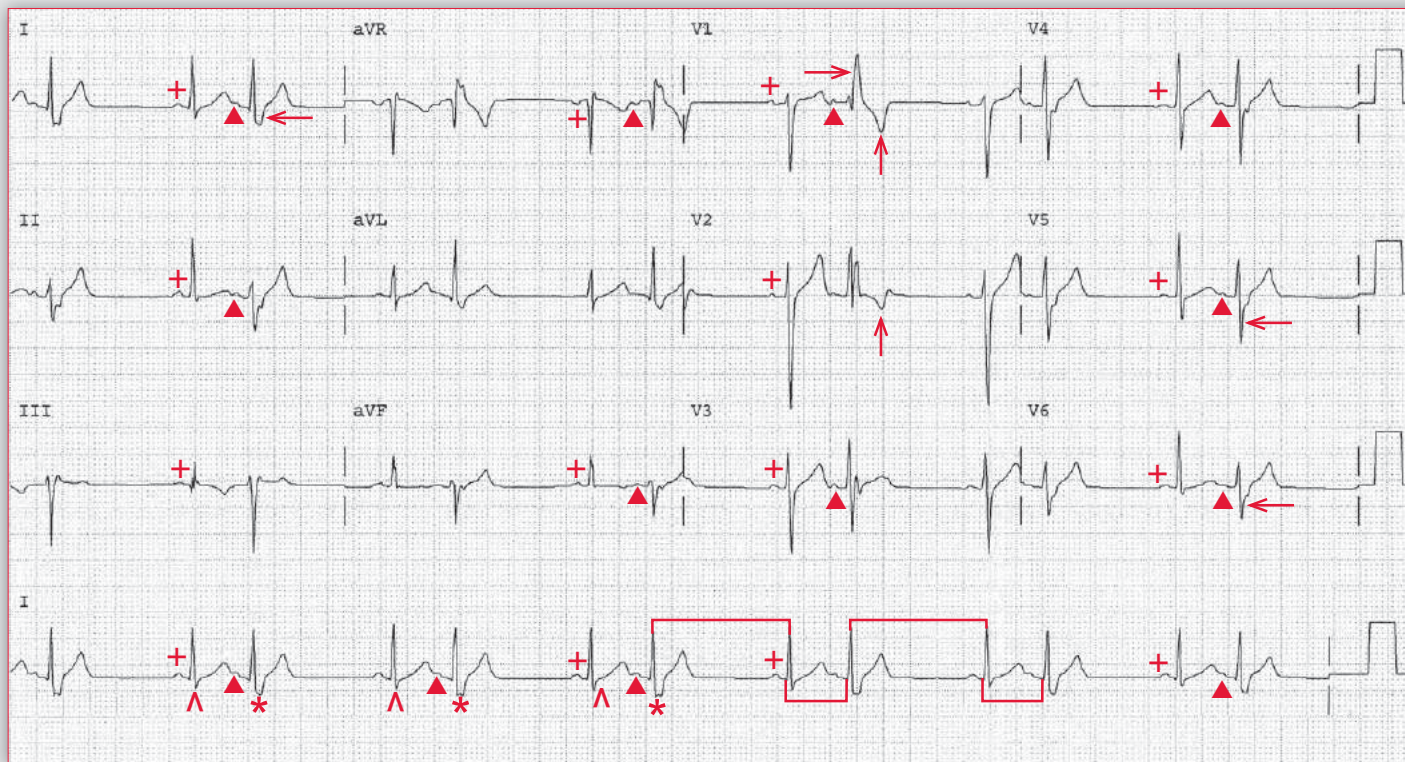
You are consulted because of an abnormal post-operative ECG in an asymptomatic patient who underwent recent cholecystectomy. The surgical team is concerned about heart block given the “grouped” beating pattern on ECG and the patient’s β -blocker has been held.

What is seen on the ECG?

What would you recommend to the team?



Podrid's Real-World ECGs



ECG 83 Analysis: Normal sinus rhythm, premature atrial complexes in a bigeminal pattern (atrial bigeminy), rate-related or functional right bundle branch block

ECG 83 shows a regularly irregular rhythm with long (\sqcap) and short (\sqcup) RR intervals. In addition, there are narrow (\wedge) and wide (\ast) QRS complexes. The average rate is 82 bpm. There is a P wave before each narrow QRS complex ($+$) and the PR interval is stable (0.14 sec). The P wave is upright in leads I, II, aVF, and V4–V6. Hence there is a normal sinus rhythm. The QRS morphology is normal and there is a normal axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). After each sinus complex, there is a premature QRS complex (\ast) that is preceded by a P wave (\blacktriangle) with a morphology that is slightly different from the sinus P wave. In addition, the PR interval is slightly longer (0.16 sec). These are premature atrial complexes in a bigeminal pattern (*ie*, every QRS complex is a premature atrial complex). The premature atrial complex has a QRS duration that is increased (0.16 sec) and it has a morphology of a typical right bundle branch block with RSR' in lead V1 (\rightarrow) and broad S waves in leads I and V4–V6 (\leftarrow). Therefore, the premature complexes have a rate-related or a functional right bundle branch block aberration. Associated with the right bundle

branch block are T-wave inversions in leads V1–V2 (\uparrow) that are repolarization abnormalities secondary to the right bundle branch block. The QT/QTc intervals are normal (320/375 msec).

Premature atrial complexes are typically followed by a pause, as observed in this ECG. Hence there is an appearance of group beating. There is no evidence of heart block and, therefore, there is no reason to withhold this patient's β -blocker. Premature atrial complexes are common, seen in about 90% of the population. They do not require therapy. However, they may be associated with symptoms, such as a sense of palpitations.

The premature atrial complexes have a right bundle branch block and this is rate-related, *ie*, the premature atrial complexes occurs at a more rapid rate than the sinus rate and as a result of underlying conduction abnormality of the right bundle, the impulse cannot be conducted through the right bundle when a certain rate occurs. ■

Notes

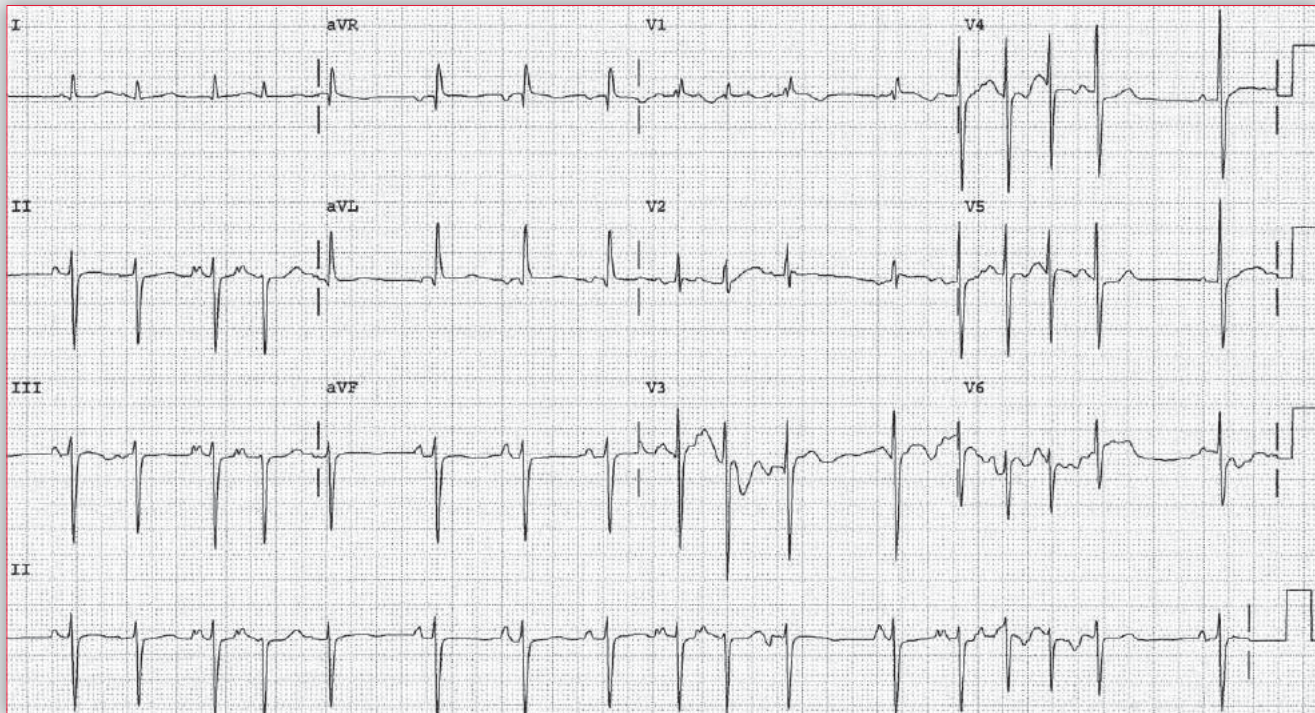
Practice Case 84

A 36-year-old woman with scleroderma presents with two months of exertional dyspnea and new-onset palpitations. The following ECG is obtained.

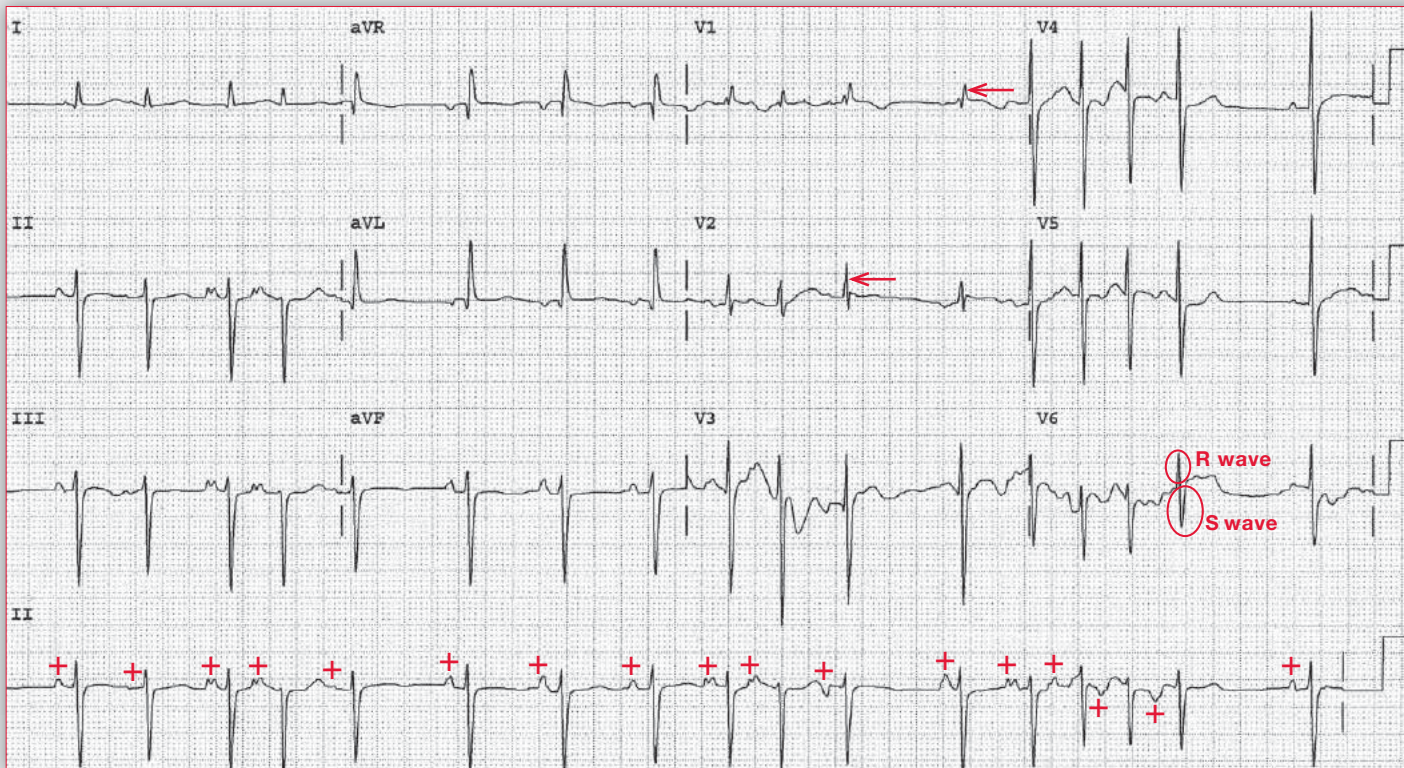
What is the rhythm abnormality?

What clinical disorder is suggested by the ECG?

How would you further evaluate this patient?



Podrid's Real-World ECGs



ECG 84 Analysis: Multifocal atrial tachycardia, left anterior fascicular block, possible right ventricular hypertrophy

ECG 84 shows an irregularly rhythm without any pattern to the RR intervals; hence the rhythm is irregularly irregular. The average rate is 108 bpm. There are only three supraventricular rhythms that are associated with an irregularly irregular rhythm. They include sinus arrhythmia, in which there is one P-wave morphology and one PR interval; multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm), in which there are > 3 different P-wave morphologies with any dominant P-wave morphology and variable PR intervals; or atrial fibrillation, in which there are no organized P waves. There are P waves before each QRS complex (+), but they have multiple morphologies (> 3 different P-wave morphologies) and there is no dominant P-wave morphology. In addition, there are variable and not constant PR intervals. These features (irregularly irregular rhythm at a rate > 100 bpm, variable P-wave morphologies and variable PR intervals) are consistent with a multifocal atrial tachycardia. The QRS complex duration is normal (0.08 sec) and there is an extremely leftward axis leftward between -30° and -90° (positive in lead I and negative in leads II and aVF with an rS morphology). This is a left anterior fascicular block. There are prominent R waves in leads V1–V2 (\leftarrow). Etiologies for a tall R wave in lead V1 include right ventricular hypertrophy (usually associated with right axis and right atrial abnormality), posterior wall myocardial infarction (often associated with an inferior wall myocardial infarction), Duchenne's muscular dystrophy (associated with a posterolateral infarction pattern), Wolff-Parkinson-White pattern (associated with a short PR interval and prolonged QRS complex duration), dextrocardia

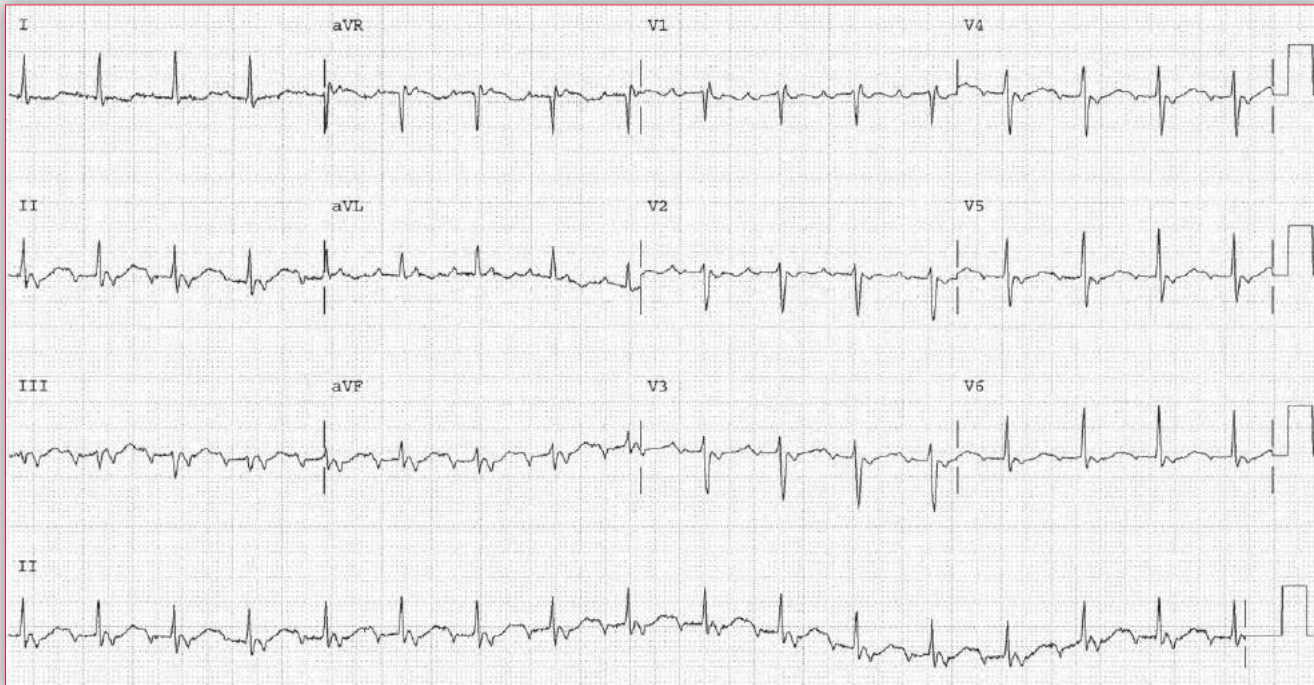
(associated with a right axis and negative P waves in leads I and aVL, and reverse R wave progression across the precordium), V1–V3 lead misposition, right-sided leads (with reverse R-wave progression across the precordium), cardiomyopathy (associated with prominent septal Q waves), and counterclockwise rotation. In this case, there is also an R/S ratio < 1 in leads V5–V6, which along with the tall R wave in leads V1 is suggestive of right ventricular hypertrophy, particularly since there are no other findings on the ECG consistent with other causes for the tall R wave in lead V1. The QT/QTc intervals are normal (320/430 msec).

Multifocal atrial tachycardia (when the rate is > 100 bpm) and multifocal atrial rhythm or wandering atrial pacemaker (when the rate is < 100 bpm) are often associated with the presence of underlying lung disease. The constellation of findings on this ECG (*ie*, right ventricular hypertrophy and multifocal atrial tachycardia) and the presenting symptoms are concerning for significant pulmonary disease and pulmonary arterial hypertension secondary to scleroderma. Initial evaluation of the patient would include a transthoracic echocardiogram to evaluate right-sided pressures, *ie*, pulmonary artery and right ventricular pressures and right ventricular function. The finding of a pulmonary artery systolic pressure > 40 mm Hg on echocardiogram suggests pulmonary artery systolic hypertension, and this is usually confirmed with a right heart catheterization and the finding of a mean pulmonary artery pressure > 25 mm Hg. ■

Practice Case 85

A 68-year-old man with chronic obstructive pulmonary disease presents with acute dyspnea, wheezing, and palpitations. He has no known cardiac disease. The following is his ECG.

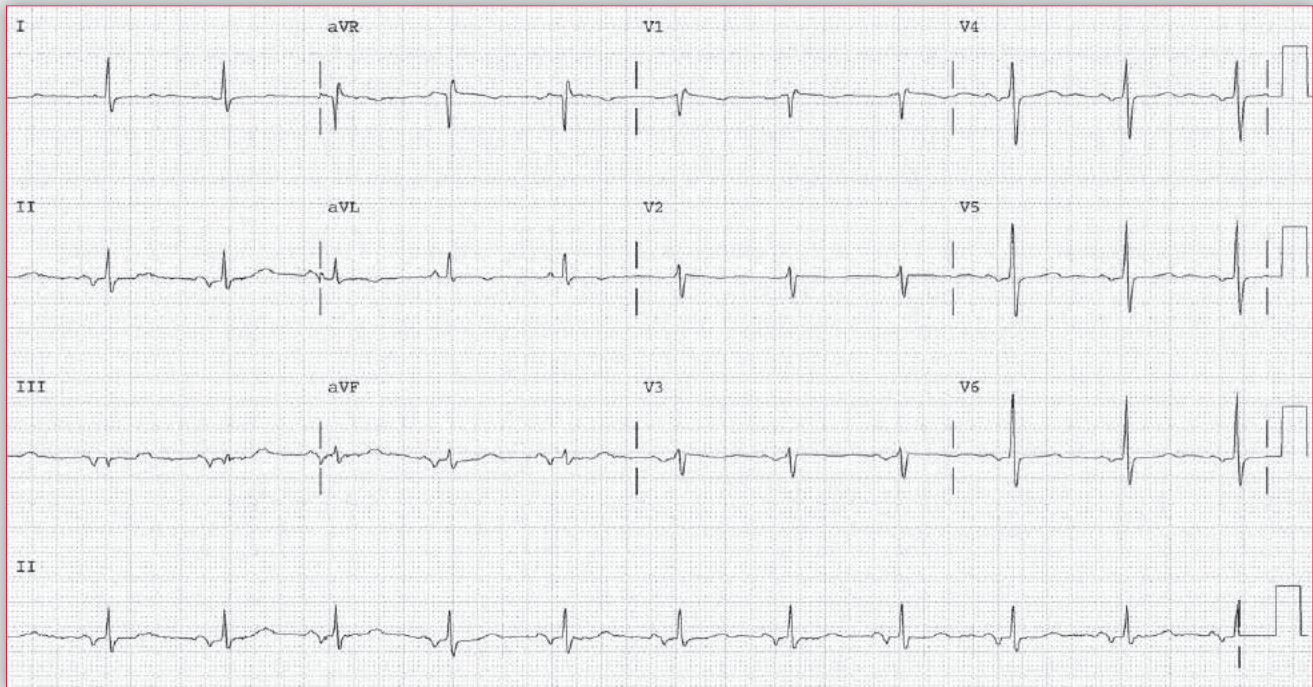
ECG 85A



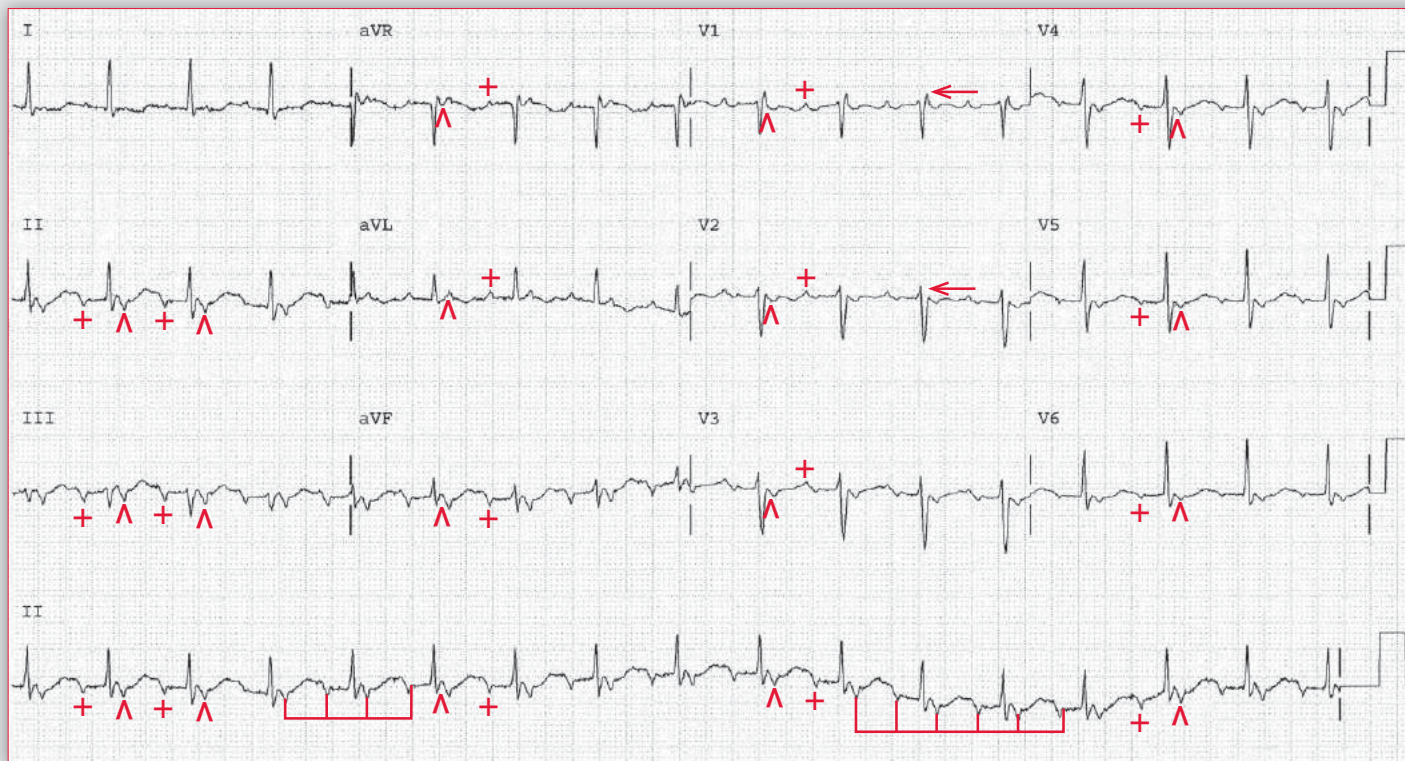
Practice Case 85

What is the rhythm abnormality?

ECG 85B



Podrid's Real-World ECGs



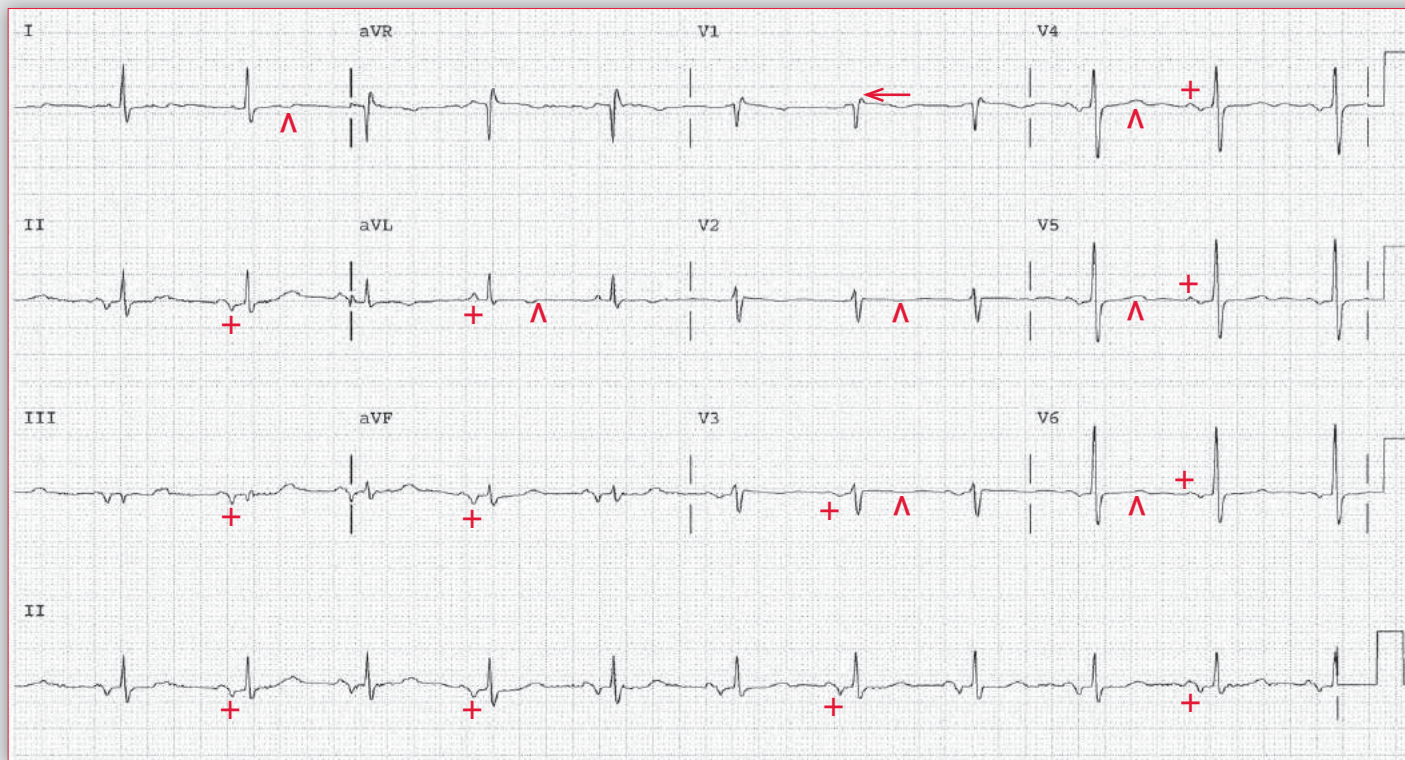
ECG 85A Analysis: Ectopic atrial tachycardia with 2:1 AV block

ECG 85A shows a regular rhythm at a rate of 100 bpm. The QRS complex duration is normal (0.08 sec), and the axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QRS complex morphology is normal, but there is an R' seen in leads V1–V2 (\leftarrow), suggesting a right ventricular conduction delay. There is a P wave (+) seen before each QRS complex with a stable PR interval (0.24 sec). The P wave is negative in leads II and aVF. Hence the atrial activity is not sinus but is from an ectopic atrial focus. A second, similar waveform (^)

can be seen immediately after the QRS complex within the ST segment. The interval of these atrial waveforms is regular (\sqcup) and hence there is a regular atrial rate of 200 bpm. The rhythm is atrial tachycardia with 2:1 AV block. Having identified a second P wave immediately after the QRS complex, it can now be seen that the R' in leads V1 and V2 (\leftarrow) is actually the second atrial waveform, and not only a right ventricular conduction delay. As a result of a P wave superimposed on the end of the T wave it is difficult to establish the QT/QTc intervals.

continues

Podrid's Real-World ECGs



ECG 85B Analysis: Ectopic atrial rhythm, diffuse nonspecific ST-T wave abnormalities

ECG 85B is from the same patient as ECG 85A. There is a regular rhythm at a rate of 68 bpm. The QRS complex duration, morphology, and axis are identical to what is seen in ECG 85A. There is an R' in V1 (\leftarrow), although it is less prominent than in ECG 85A. On ECG 85A there was a P wave superimposed on the R', augmenting its amplitude. The QT/QTc intervals are normal (420/440 msec).

There is a P wave (+) before each QRS complex with a stable PR interval (0.24 sec), which is the same as the PR interval seen in ECG 85A. The P wave is negative in leads II and aVF and biphasic in leads V4–V6. It has a similar morphology to the P wave seen in ECG 85A. Hence this

is an ectopic atrial rhythm, while on ECG 85A the rate of the ectopic atrial rhythm faster (*ie*, 200 bpm), and as the rate is > 100 bpm this is an atrial tachycardia. Also noted are nonspecific ST-T wave abnormalities in leads I, aVL, and V2–V6 (\wedge).

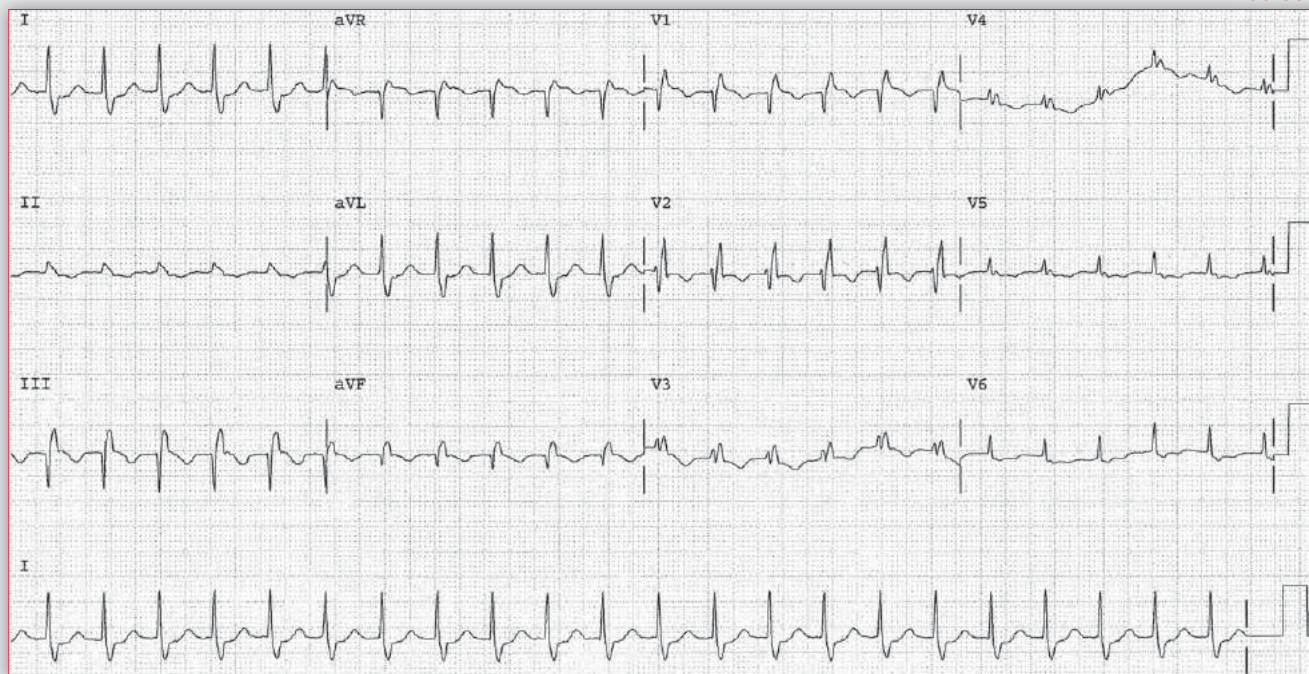
Atrial arrhythmias are commonly seen in association with pulmonary disease. Initial therapy is generally rate control using an AV nodal blocking agent, *ie*, a β -blocker, calcium-channel blocker, or digoxin. Often the atrial arrhythmia spontaneous reverts to sinus rhythm with treatment of the underlying pulmonary condition. If it persists, therapy with a class IA, IC, or III antiarrhythmic agent can be initiated. ■

Practice Case 86

A 69-year-old woman with known coronary disease presents to the emergency department with sudden-onset lightheadedness and palpitations. An ECG is obtained (ECG 86A). Except for the rapid heart rate, her physical examination is otherwise normal. Intravenous

access is established and a medication is administered, and there is an abrupt termination of the tachycardia. The symptoms also resolve with the reversion of the tachycardia. A follow-up ECG is obtained (ECG 86B).

ECG 86A

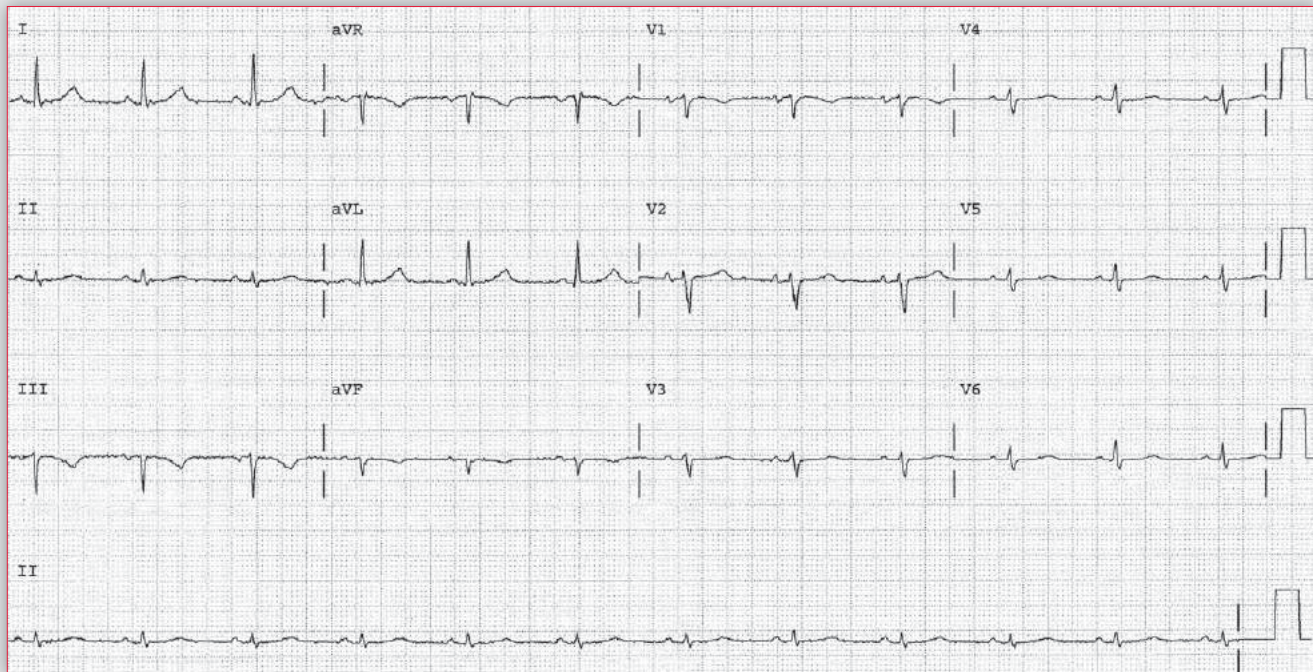


Practice Case 86

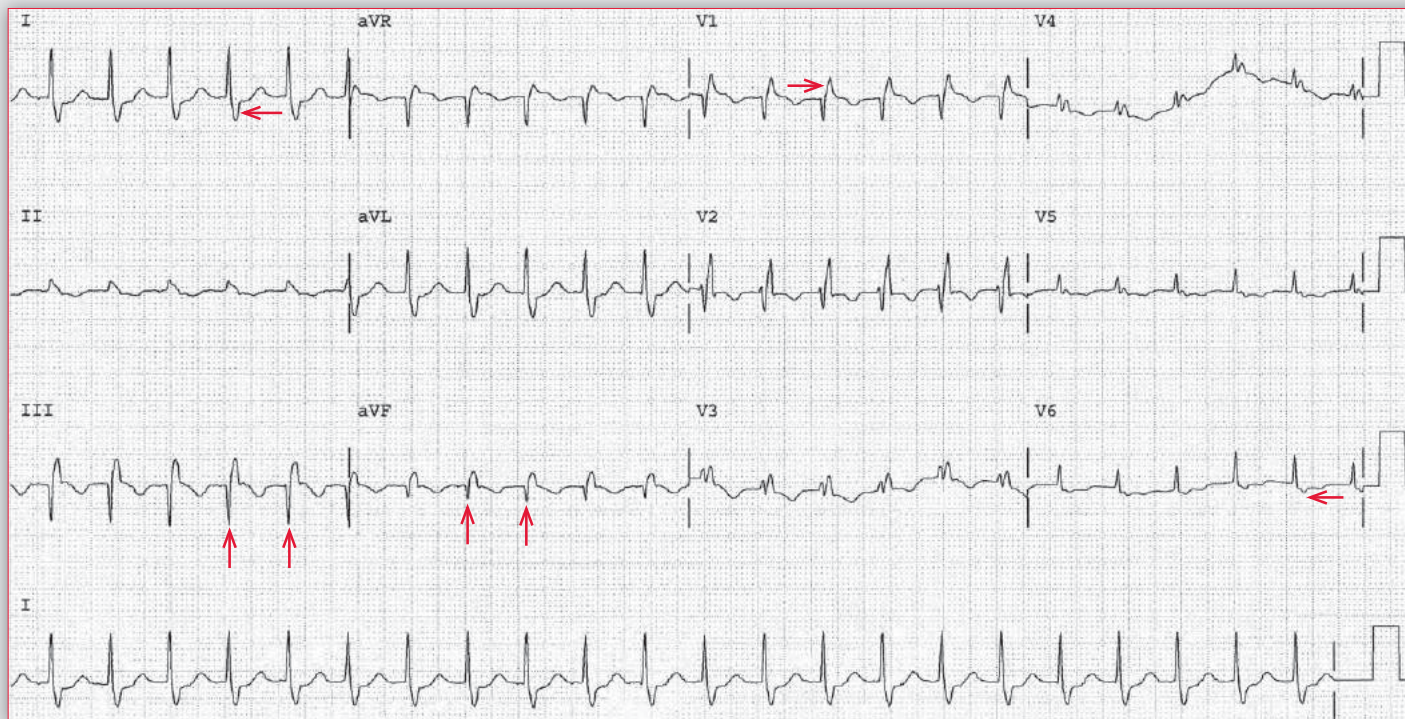
What is the most likely mechanism of the tachyarrhythmia?

How did the ED attending acutely treat this patient?

ECG 86B



Podrid's Real-World ECGs



ECG 86A Analysis: Narrow complex, no-RP supraventricular tachycardia, atrioventricular nodal reentrant tachycardia, rate-related right bundle branch block, left axis, old inferior myocardial infarction, low voltage in the precordial leads

ECG 86A is a regular rhythm at a rate 136 bpm. There are no P waves seen before or after any QRS complex. The QRS complex duration is prolonged (0.14 sec), and there is a right bundle branch block pattern with an RSR' in V1 (→) and broad S wave in leads I and V6 (←). The axis is leftward between 0° and −30° (positive QRS complex in leads I and II and negative in lead aVF). There are Q waves in leads III and aVF (†), indicative of an inferior wall myocardial infarction, which is the etiology of the leftward axis. The QT/QTc intervals are prolonged (320/480 msec), but they are normal when the prolonged QRS complex duration is considered (280/410 msec). There is low voltage in the precordial leads (< 10 mm or little boxes in each lead).

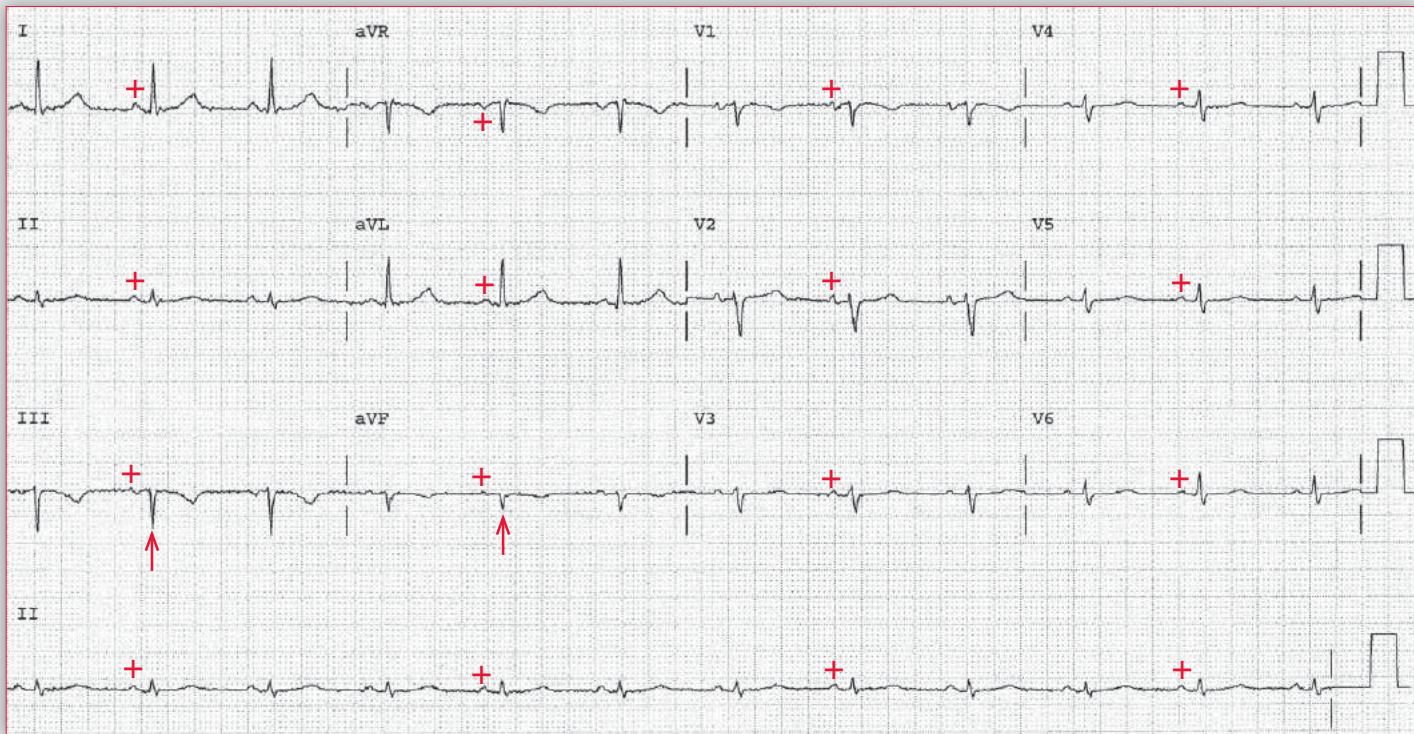
This is a supraventricular tachycardia, and in the absence of any definitive P waves, it is termed a no-RP tachycardia. The most likely etiology for a no-RP tachycardia is a typical atrioventricular reentrant tachycardia (AVNRT) due to dual AV nodal pathways. There is a fast pathway, which conducts rapidly but has a long refractory period and recovers slowly and a slow pathway, which conducts the impulse slowly but has a short refractory period and recovers quickly. These two pathway a linked proximally distally in the AV node, forming a circuit. In sinus

rhythm, the impulse to the ventricles is via the fast pathway, and the slow pathway is not active. However, if there is a premature atrial complex that arrives at the AV node before the fast pathway has recovered, the impulse will be conducted antegradely to the ventricles via the slow pathway (and hence associated with a long PR interval). If the impulse arrives at the bundle of His when the fast pathway has recovered, the impulse will enter the fast pathway to be conducted rapidly in a retrograde direction back to the atria. Thus, there will be simultaneous activation of the ventricles and atria, resulting in the absence of an obvious P wave. If the slow pathway has recovered when the impulse reaches the atria, the impulse can reenter the slow pathway and the process will repeat itself, resulting in a reentrant arrhythmia, *ie*, an AVNRT that is called slow-fast.

Acutely, AVNRT can be terminated by any agent that affects AV nodal electrophysiologic properties. This includes IV adenosine, a β -blocker (administered either orally or IV), a calcium-channel blocker (administered orally or IV) or digoxin. The chronic treatment of AVNRT includes β -blockers, calcium-channel blockers, digoxin, or radio-frequency ablation.

continues

Podrid's Real-World ECGs



ECG 86B Analysis: Normal sinus rhythm, left axis, old inferior myocardial infarction, low-voltage in the precordial leads

ECG 86B is from the same patient as ECG 86A. There is a regular rhythm at a rate of 66 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. The QRS complex duration is normal (0.08 sec) and there is a normal morphology, although the QRS voltage is low in the precordial leads (< 10 mm or little boxes in each lead). The axis is leftward between 0° and –30° (positive QRS complex in leads I

and II and negative in lead aVF). The QT/QTc intervals are normal (400/425 msec). There are Q waves in leads III and aVF (↑), indicative of an inferior wall myocardial infarction, which is the etiology of the leftward axis. This is similar to what was seen in ECG 86A. However, a right bundle branch block morphology is not seen, and this is due to the fact that the rate is slower. Hence ECG 86A shows an AVNRT with a rate-related right bundle branch block. ■

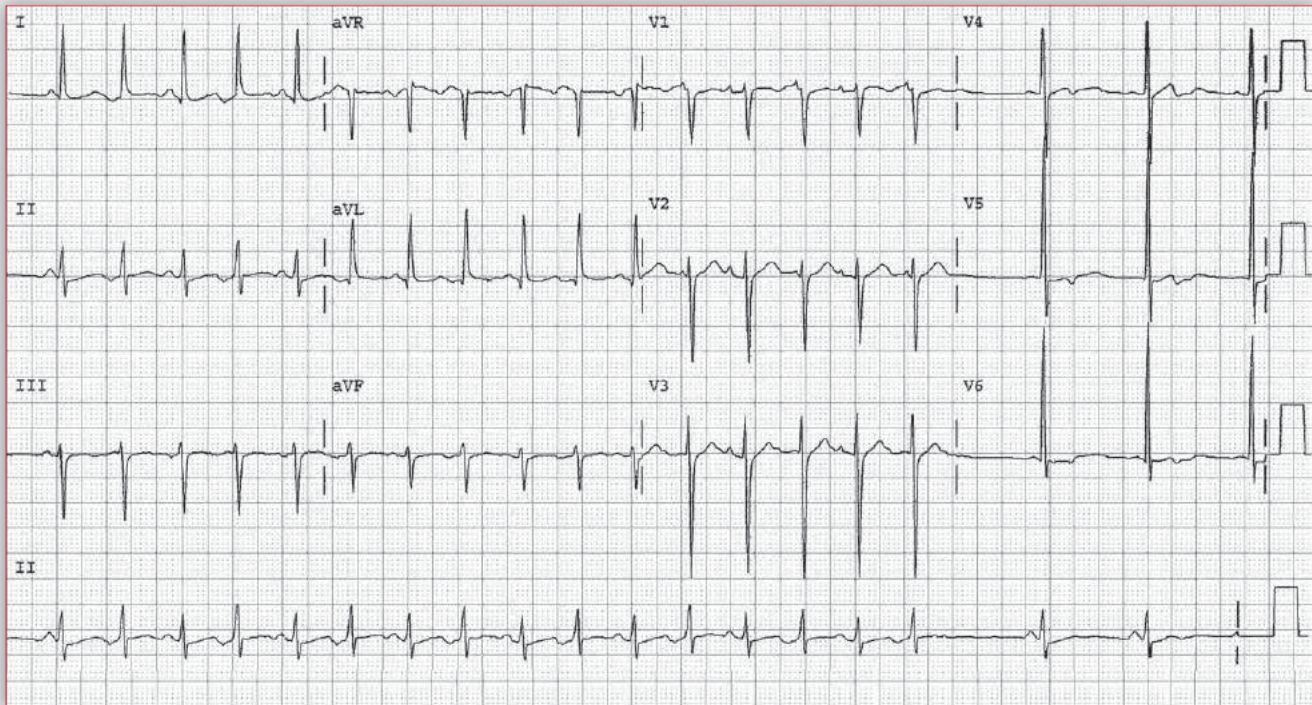
Notes

Practice Case 87

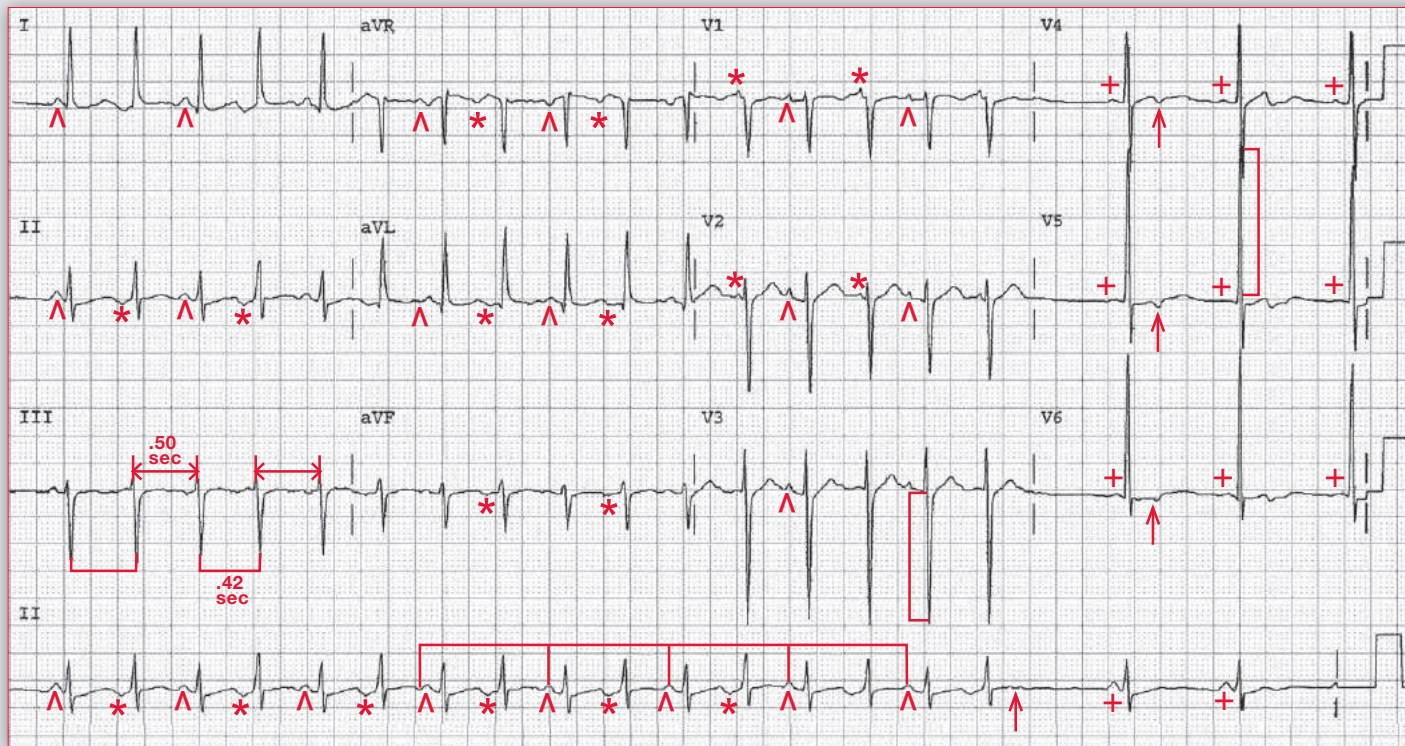
The following ECG is obtained from a 34-year-old healthy woman with intermittent sensation of an irregular heartbeat. She has no known cardiovascular or pulmonary disease. The patient's PCP diagnoses either atrial flutter or atrial tachycardia.

Do you agree with this diagnosis?

What therapy should be considered?



Podrid's Real-World ECGs



ECG 87 Analysis: Normal sinus rhythm, interpolated premature atrial complexes in bigeminal pattern (atrial bigeminy), left ventricular hypertrophy with associated ST-T wave abnormalities, left axis

ECG 87 shows that there appears to be a regular rhythm at a rate of 140 bpm. However, it can be seen that there are two different RR intervals, longer (duration 0.50 sec) (\leftrightarrow) and shorter (0.42 sec) (\sqcup). The QRS complex duration is normal (0.08 sec), and there is a leftward axis between 0° and -30° (positive QRS complex in leads I and II and negative in lead aVF). The QRS complex morphology is normal, but there is tall R wave in lead V5 (28 mm) (\sqcup) and a deep S wave in lead V3 (25 mm) (\sqcup), for a total of 53 mm, which meets one of the diagnostic for left ventricular hypertrophy (*ie*, R-wave voltage in lead V5 + S wave voltage in lead V3 > 35 mm). There are also ST-T wave changes seen in leads V4–V6 (\uparrow), which are probably associated with left ventricular hypertrophy. At the end of the tracing, there is a long RR interval with an abrupt slowing of the rate to 64 bpm. This appears to be the result of a nonconducted premature P wave (\uparrow), which is seen as an irregularity of the ST segment (in lead II rhythm strip), but is not seen with other QRS complexes. The QT/QTc intervals are normal (280/430 msec).

There is a clear P wave before the last three QRS complexes (+) with a stable PR interval (0.14 sec). Therefore, these are sinus complexes at a rate of 64 bpm. A P wave with the same morphology can be seen before every other QRS complex in the beginning part of the tracing (\wedge), *ie*, the QRS complex associated with the longer RR interval. These P waves have the same PP interval (rate 64 bpm) as the PP intervals at the end (\sqcap). The P waves are positive in leads I, II, aVF, and V4–V6. Hence

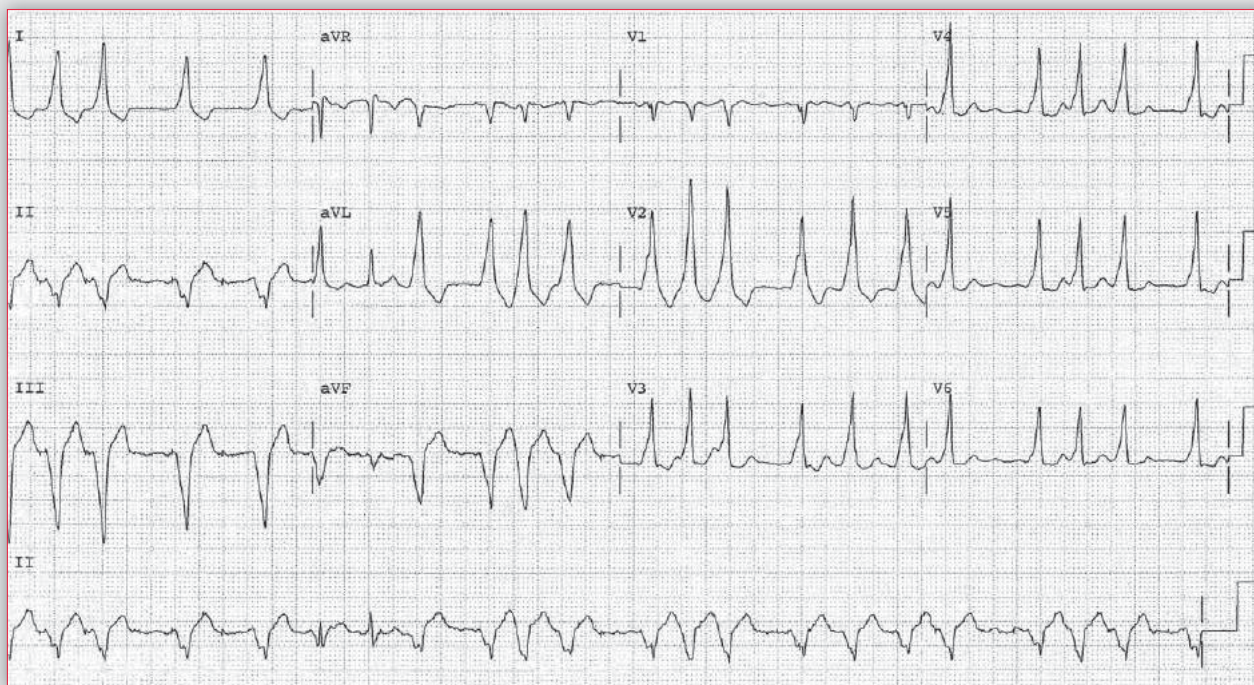
these are sinus complexes. There is also a P wave (*) before the alternating QRS complexes associated with the shorter RR interval; however these P waves are negative in leads I, II, and aVF. They are associated with a shorter PR interval (0.12 sec) compared to the sinus complex. Hence they are not sinus P waves, but are from an ectopic atrial focus. Hence this is a sinus rhythm with premature atrial complexes in a bigeminal pattern. This accounts for the slightly shorter RR interval (\sqcup). Since these complexes do not alter the PP interval (\sqcap), the premature atrial complexes are termed “interpolated.” Therefore, this is not a supraventricular tachyarrhythmia, as the sinus rate is actually 64 bpm and the rapid rate is the result of atrial bigeminy.

The rhythm is not regular, as there is a pattern of longer and shorter RR intervals, which is the result of atrial bigeminy. Hence this is not an atrioventricular nodal reentrant tachycardia. In addition, there are two distinctly different P waves, which are seen in an alternating pattern. Therefore, this is not an ectopic atrial tachycardia in which only one P-wave morphology would be seen. Moreover, as there is only one P wave before each QRS complex, without evidence of other atrial activity, this is not atrial flutter. Although there are two different P-wave morphologies occurring in an alternating fashion, this is not multifocal atrial tachycardia (which requires ≥ 3 different P-wave morphologies without a dominant P-wave morphology). ■

Practice Case 88

A 22-year-old college student-athlete presents to the health service because of a pre-syncope episode. He is still complaining of palpitations. An ECG is obtained (ECG 88A). The health service has a copy of his baseline ECG, which was obtained prior to his joining the college basketball team (ECG 88B).

ECG 88A

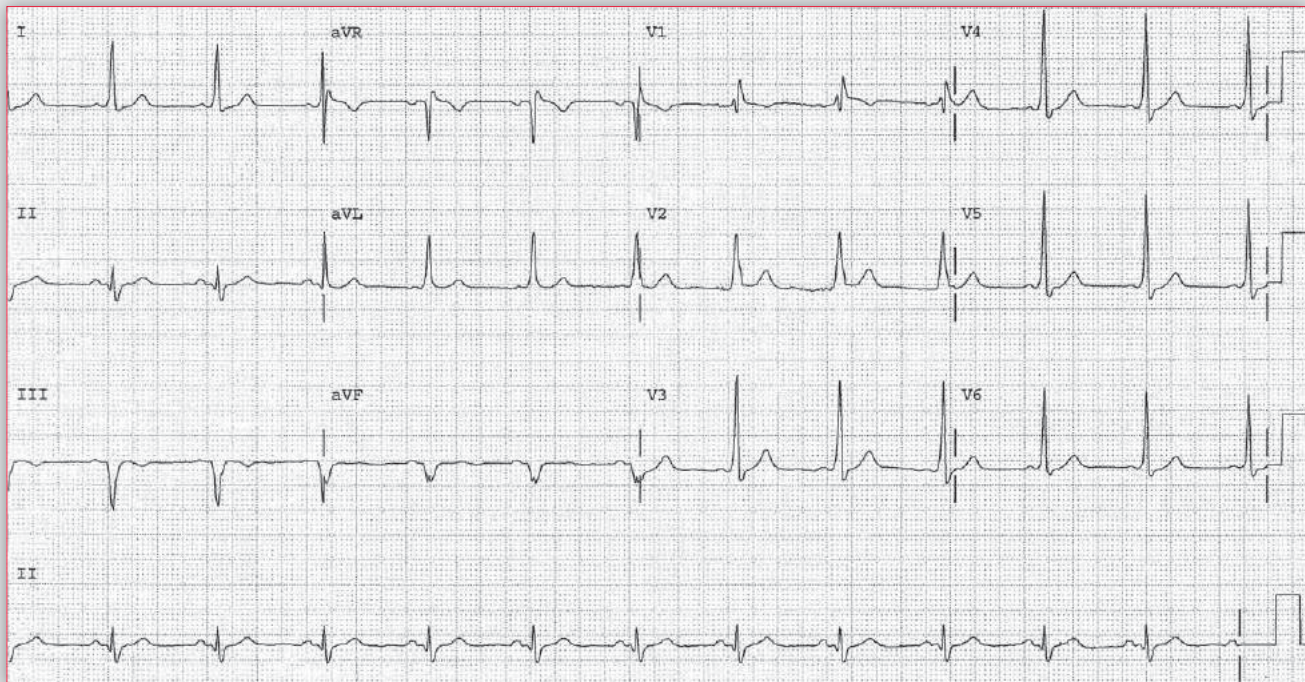


Practice Case 88

What is the diagnosis?

What would you recommend for first-line acute treatment?

ECG 88B





ECG 88A Analysis: Atrial fibrillation, Wolff-Parkinson-White (WPW) pattern

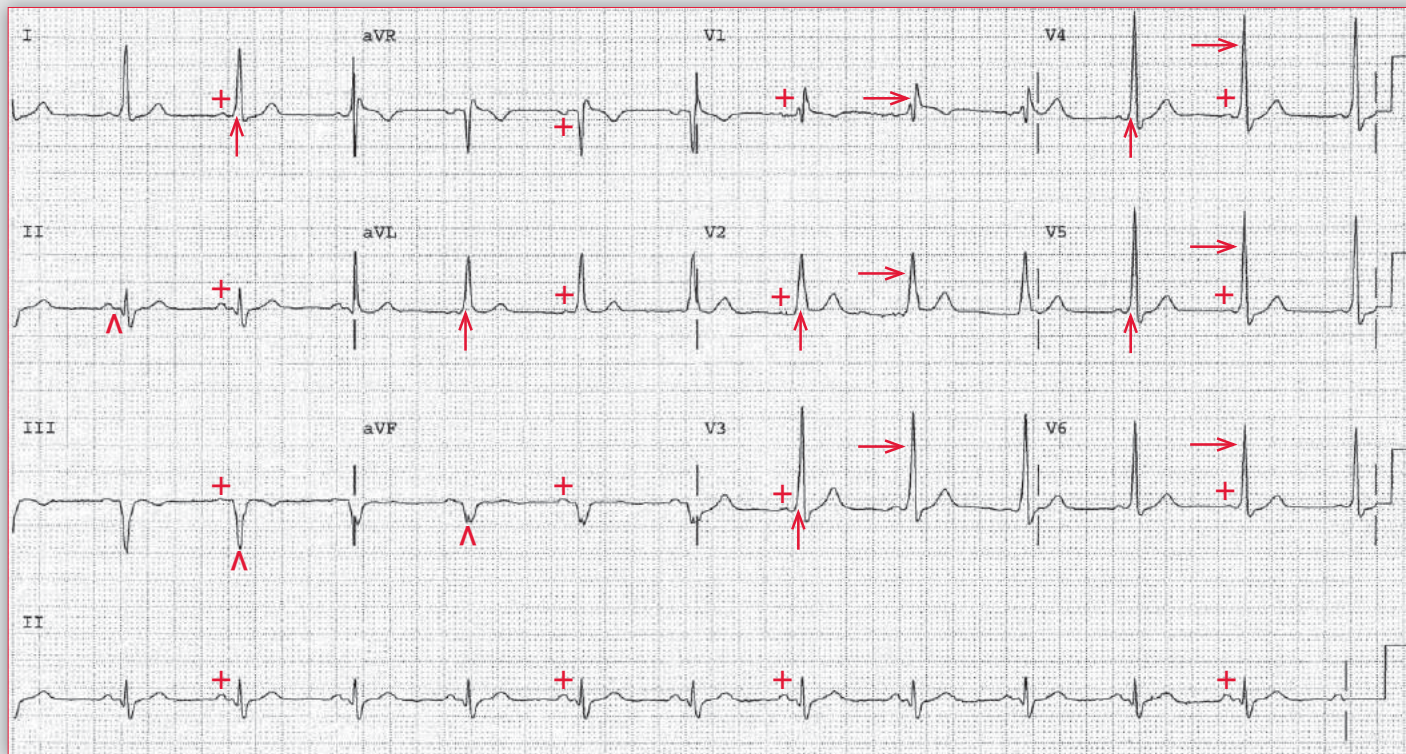
ECG 88A shows the rhythm is irregularly irregular, with an average rate of 132 bpm. There are only three supraventricular rhythms that are associated with an irregularly irregular rhythm. They include sinus arrhythmia, in which there is one P-wave morphology and one PR interval; multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm), in which there are > 3 different P-wave morphologies with no P-wave morphology being dominant and variable PR intervals; or atrial fibrillation, in which there are no organized P waves. There are no obvious P waves seen, but there is evidence of rapid and irregular atrial activity, especially in leads II and V1 (^). The underlying rhythm is thus atrial fibrillation. The QRS complexes have variable durations and morphologies. The sixth QRS complex (*) has a normal duration (0.08 sec) and in leads II, aVR, aVL, and aVF appears to have a normal morphology. The fifth QRS complex (●) has a similar morphology, although it is slightly wider (0.12 sec). All the other QRS complexes (+) are wide (0.16 sec) and there are subtle differences in their width. This appears to be due to differences in the duration of the upstroke of the QRS complex, as can be best seen in lead V4 (†). Importantly, there is no relationship between the QRS complex width and the RR intervals. Hence, there are narrow QRS complexes following a short RR interval (□), *ie*, faster rate (for example the fifth and sixth QRS complexes), while there are wider QRS complexes that follow a long RR interval (⊐) or faster rate, for example complexes 8 and 11 (▲). Usually aberration is rate-related, *ie*, the QRS complex is wider at a faster rate. The absence of an association between rate (RR interval) and QRS complex width is seen with pre-excitation, specifically in Wolff-Parkinson-White. Supporting this is the non-rate-related variability of the width of the wide QRS complexes. This represents various

degrees of fusion between conduction via the accessory pathway and the normal AV node–His–Purkinje system. Therefore, this is atrial fibrillation associated with Wolff-Parkinson-White syndrome. The slowed or slurred upstroke is a delta wave, and the variation in the delta wave width reflects variability in how much of the left ventricular myocardium is activated via the accessory pathway. This reflects the balance of conduction through the accessory pathway and the normal AV node–His–Purkinje system. If AV nodal conduction is slow, more of ventricular myocardial activation occurs via the accessory pathway, and hence the delta wave is more prominent or wider. If AV nodal conduction is faster, less ventricular myocardium is activated via the accessory pathway and the delta wave is, therefore, narrower and less prominent. These changes are not related to the ventricular rate.

ECG 88B is the baseline ECG for ECG 88A. There is a regular rhythm at a rate of 72 bpm. There is a P wave before each QRS complex (+) with a stable, but short PR interval (0.12 sec). The QRS complex duration is prolonged (0.14 sec) as a result of a slow, slurred upstroke of the QRS complex (†), which is a delta wave, reflecting early and slowed myocardial activation initiated by an impulse via the accessory pathway. This is a Wolff-Parkinson-White pattern. The remainder of the QRS complex is narrow, a result of subsequent myocardial activation via the normal AV node–His–Purkinje pathway. Hence, the QRS complex in Wolff-Parkinson-White pattern represents fusion of activation via both of these pathways that link the atria with the ventricles. Also noted is positive concordance, *ie*, tall R waves from V1–V6 (→). Positive concordance may be seen when there is direct activation of the

continues

Podrid's Real-World ECGs



ECG 88B Analysis: Sinus rhythm, Wolff-Parkinson-White pattern

ventricular myocardium, including Wolff-Parkinson-White, a ventricular QRS complex, or a ventricular paced QRS complex. Also noted are Q waves in leads II, III, and aVF ^Δ. This represents a pseudo inferior wall infarction pattern, and the Q wave is the result of the delta wave. Indeed, myocardial infarction cannot be established on the ECG due to the fact that there is direct myocardial activation, with initial activation bypassing the normal conduction system. Hence, similar to the situation with a left bundle branch block, a paced complex, or a ventricular complex (all conditions associated with direct ventricular myocardial activation), a chronic myocardial infarction cannot be diagnosed on the ECG with WPW pattern. The morphology of a pseudo inferior wall myocardial infarction is associated with a posteroseptal bypass tract. The positive delta wave in lead V1 is termed type A WPW, and this is seen with a left ventricular accessory pathway in which the impulse is directed anteriorly, toward V1.

The QT/QTc intervals are normal (400/440 msec and 360/390 msec when the prolonged QRS complex duration is considered).

A specific arrhythmia resulting from WPW and requiring the accessory pathway is an atrioventricular reentrant tachycardia, due to a circuit involving the normal AV node–His–Purkinje system as one limb and the accessory pathway as the second limb. These two limbs are linked via the atria and ventricles, forming a macro-reentrant circuit. Other atrial arrhythmias, such as atrial tachycardia, atrial flutter, or atrial fibrillation, may also occur and although they are not generally due to WPW or require the accessory pathway, they may result in conduction to the ventricles via the accessory pathway. In atrial fibrillation, with atrial rates of up to 450 bpm or faster, an accessory pathway

with a short refractory period may result in rapid impulse conduction to the ventricles. In this situation, the QRS complexes will be wide and aberrated with a WPW pattern. A very rapid ventricular response rate (*ie*, > 350 to 400 bpm) during atrial fibrillation may precipitate ventricular fibrillation even in a structurally normal heart. This is the mechanism of sudden cardiac death in patients with WPW syndrome. Acute therapy of atrial fibrillation associated with WPW is cardioversion if there is hemodynamic instability. Pharmacologic therapy can be used when hemodynamics are maintained. However, AV nodal blocking agents should not be used, since blockade of the AV node will shift conduction to the ventricles to occur only via the accessory pathway. If the accessory pathway has a short refractory period, impulse conduction to the ventricle will become very rapid, and the rate acceleration can increase the potential for ventricular fibrillation. Drugs of choice for atrial fibrillation in WPW include intravenous procainamide or intravenous ibutilide. These agents not only prolong the refractoriness of the accessory pathway, slowing ventricular response and perhaps even blocking accessory pathway conduction, but they also have the potential to revert atrial fibrillation and restore normal sinus rhythm. Although not mentioned in the guidelines, lidocaine may be useful, as it will also slow conduction via the accessory pathway, resulting in narrowing and perhaps normalization of the QRS complex as conduction is shifted to the AV node–His–Purkinje system. Lidocaine will not affect the atrial fibrillation, but by blocking accessory pathway conduction, the risk of rapid ventricular rates will be reduced or eliminated. Ablation is the recommended first-line treatment in patients with atrial fibrillation and WPW to prevent a rapid ventricular response rate and the potential for ventricular fibrillation. ■

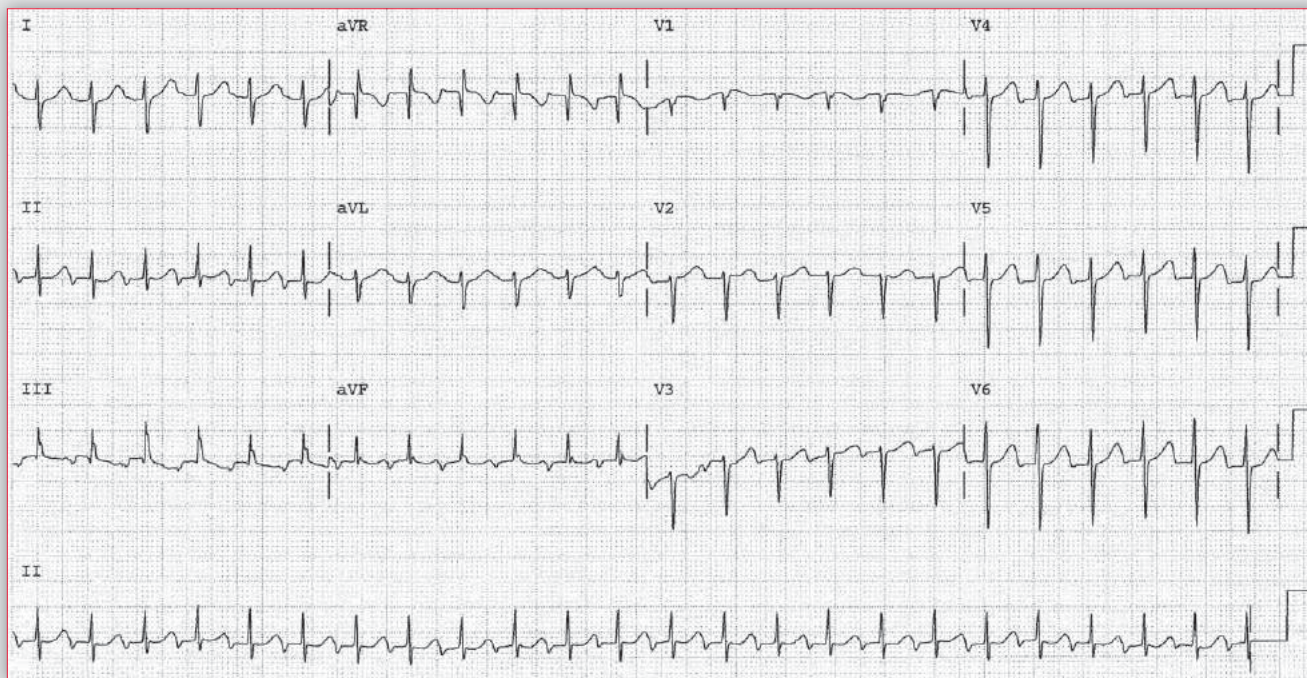
Practice Case 89

A 54-year-old diabetic with end-stage renal disease on hemodialysis presents with palpitations and shortness of breath. He states that he has missed one of his dialysis sessions. Except for a rapid heart rate and blood pressure of 150/90, his physical examination is normal.

A chest x-ray does show evidence of vascular congestion.

Dialysis is begun in the emergency department, and after about 60 minutes there is a change in his heart rate. An ECG is obtained (89B).

ECG 89A



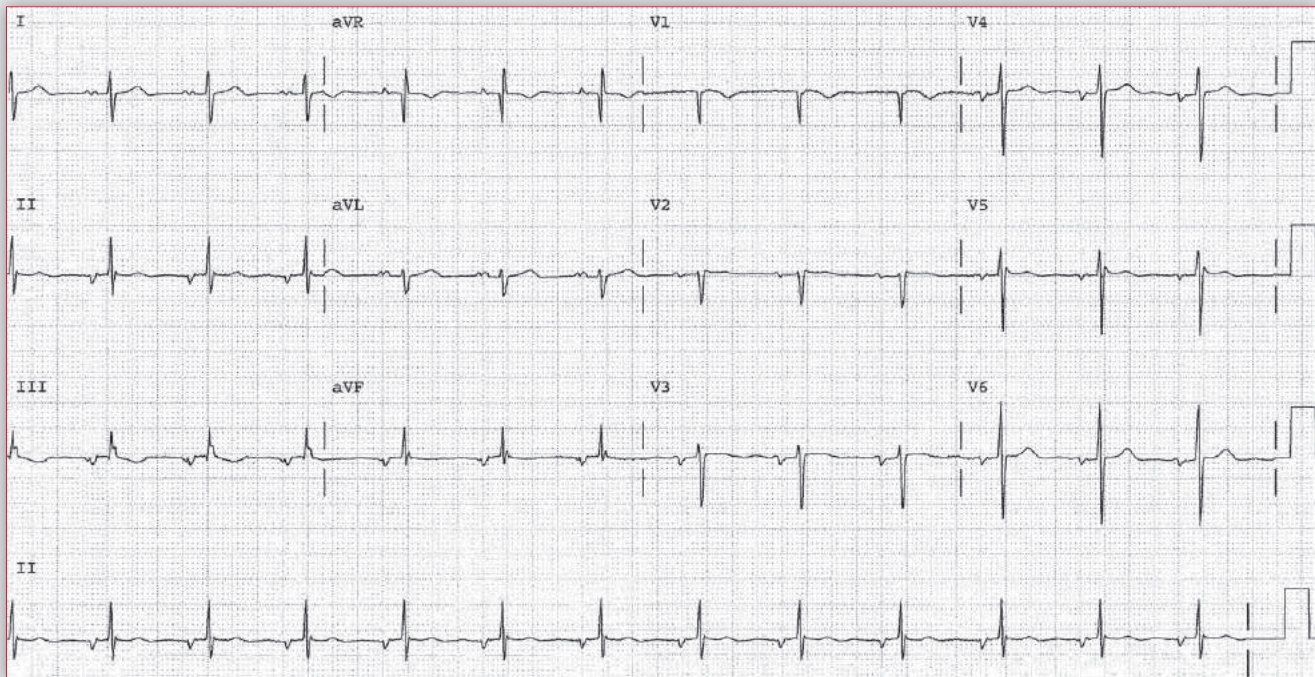
Practice Case 89

How would you classify the tachyarrhythmia in ECG 89A?

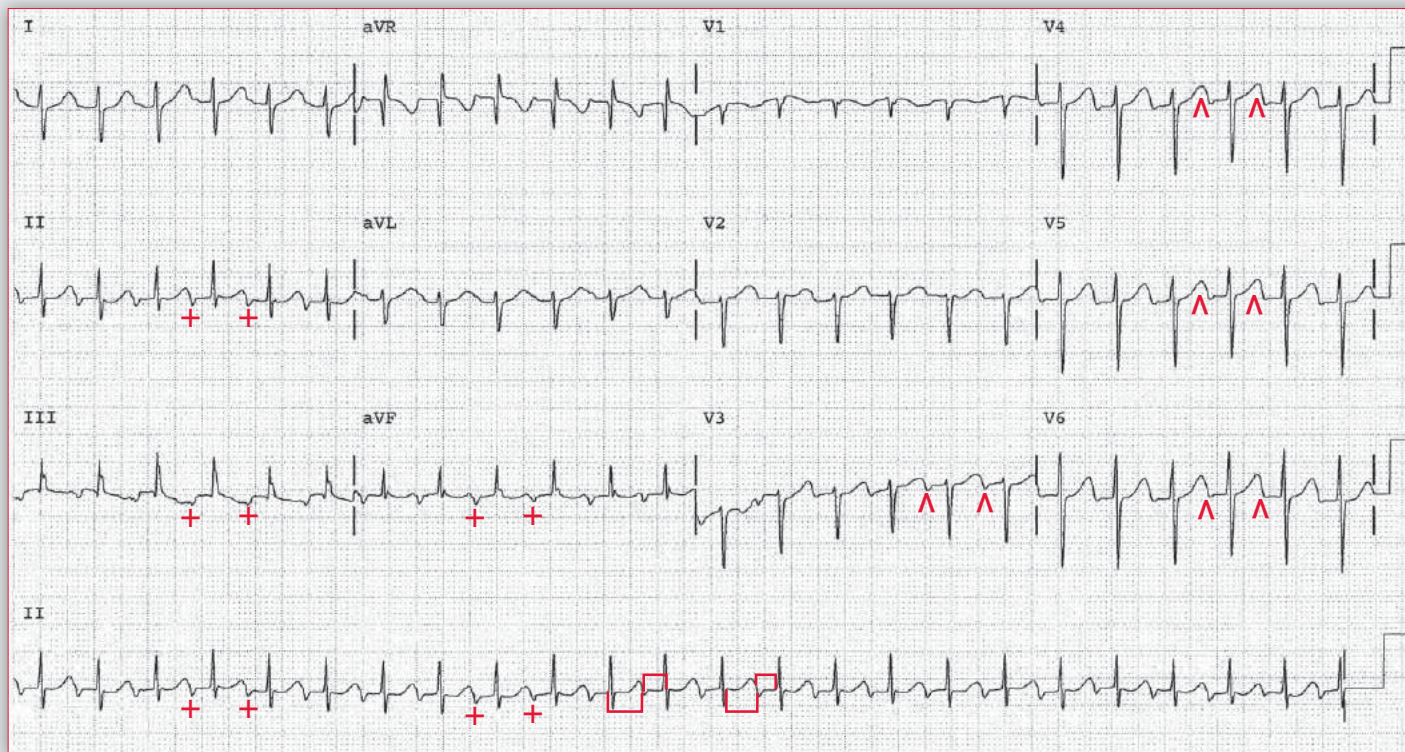
What are the different causes for this arrhythmia?

How does ECG 89B help to establish the etiology of the abnormal rhythm?

ECG 89B



Podrid's Real-World ECGs



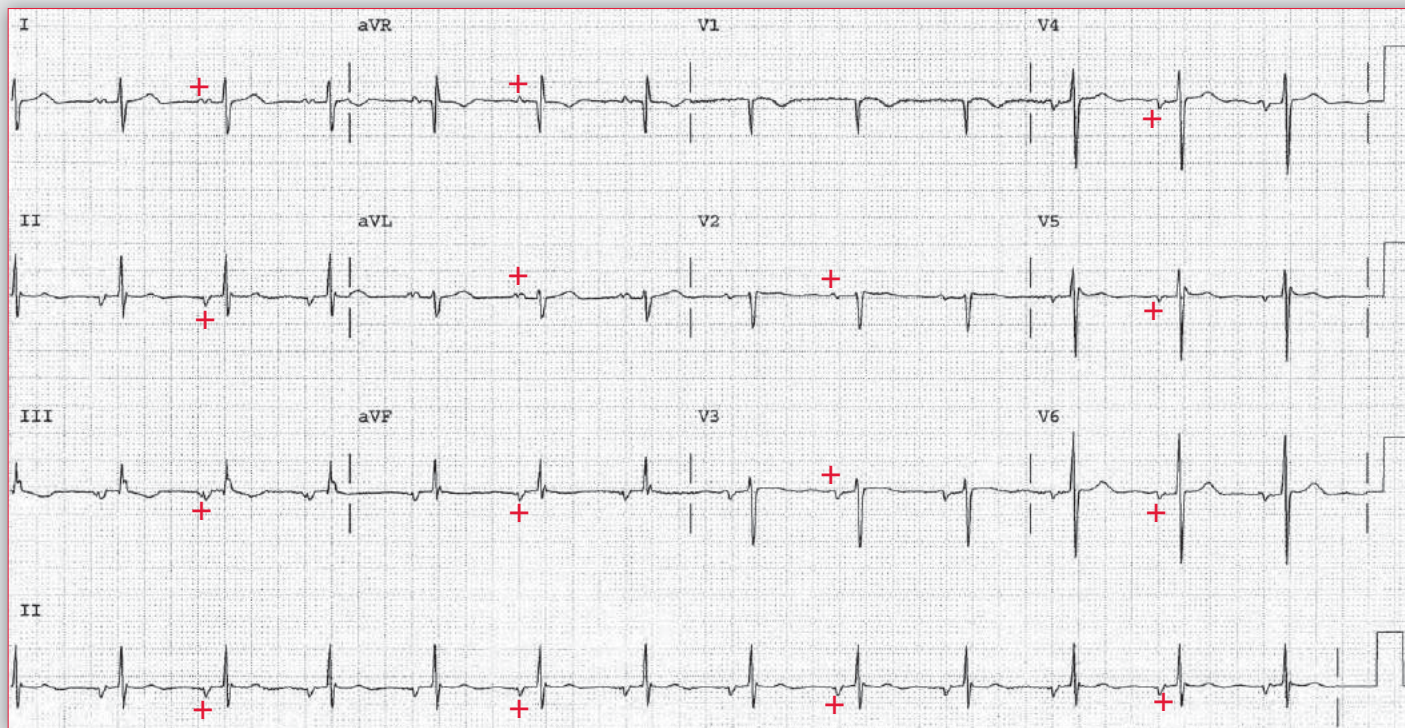
ECG 89A Analysis: Long RP supraventricular tachycardia, left posterior fascicular block, ectopic atrial tachycardia

ECG 89A shows a regular rhythm at a rate of 146 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is rightward between $+90^\circ$ and $+180^\circ$ (QRS complex negative in lead I and positive in aVF). There are several etiologies for a rightward axis including right ventricular hypertrophy (associated with a tall R wave in lead V1), lateral wall myocardial infarction (with Q wave in leads I and aVL), right-left arm lead misplacement (associated with a negative P and T wave in leads I and aVL and positive P and T wave in lead aVR), Wolff-Parkinson-White pattern (with a short PR interval and a delta wave), dextrocardia (with features of right-left arm lead switch as well as reverse R-wave progression from V1–V6), and a left posterior fascicular block. A left posterior fascicular block is a diagnosis of exclusion, *ie*, it is diagnosed only if other reasons for a rightward axis are excluded, as is the case here. The QT/QTc intervals are normal (280/440 msec).

Although there are no obvious P waves before any QRS complex, there are prominent negative waveforms seen between each QRS complex, especially in leads II, III, and aVF (+). These are in fact P waves. The RP interval (\sqcup) is 0.26 second and the PR interval (\sqcap) is 0.16 second. These intervals are constant; hence this is a long RP tachycardia. A small negative deflection, with the same PR and RP intervals, can also be seen after the T wave in leads V3–V6 (\wedge). Etiologies for a long RP tachycardia include sinus tachycardia, atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, an atypical or uncommon atrioventricular nodal reentrant tachycardia (impulse to ventricles via a fast pathway with retrograde conduction back to the atria via a slow), and atrioventricular reentrant tachycardia. The presence of a negative P wave in leads II, aVF, and V4–V6 eliminates sinus tachycardia as a cause. The absence of any evidence for a second atrial flutter wave makes this arrhythmia less likely.

continues

Podrid's Real-World ECGs



ECG 89B Analysis: Ectopic atrial rhythm, left posterior fascicular block

ECG 89B is from the same patient as ECG 89A. It was obtained after 1 hour of dialysis and the removal of almost 2 liters of fluid. There is a regular rhythm at a rate of 76 bpm. The QRS complex duration (0.08 sec), there is a normal morphology and axis (rightward) are identical to what was seen in ECG 89A. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is negative in leads II, aVF, and V4–V6. Hence this is an atrial rhythm. The P wave and PR interval are identical to those seen in ECG 89A, confirming that the rhythm upon presentation is an atrial tachycardia.

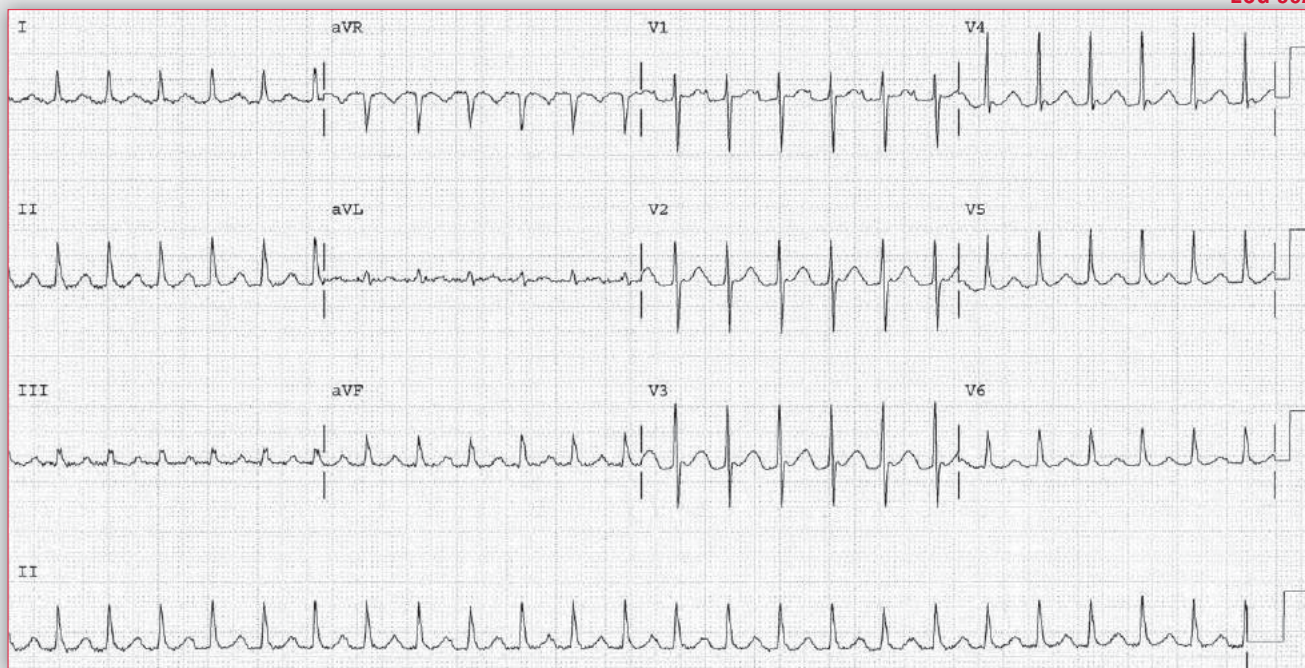
The most likely etiology for the atrial tachycardia is volume overload with the development of pulmonary vascular congestion and activation of the sympathetic nervous system that resulted in an acceleration of an underlying ectopic atrial focus. An atrial rhythm and atrial tachycardia have the same mechanism and are most often the result of an abnormal or ectopic focus. The rate of impulse generation from this ectopic focus can vary widely, and may be particularly responsive to sympathetic nervous system activation or circulating catecholamines. When the rate is < 100 bpm, it is termed an ectopic atrial rhythm, while rates of > 100 bpm define an atrial tachycardia. ■

Practice Case 90

A 64-year-old man presents to the emergency department with dizziness that has been present for the past 3 hours. He states that 2 months ago he underwent pulmonary vein isolation for symptomatic atrial fibrillation.

An ECG is obtained (ECG 90A). He is given a β -blocker as therapy to slow his heart rate. However, this therapy is ineffective, and he is admitted to a telemetry unit. After admission, he is seen by the internist, who notes that his heart rate has slowed and obtains an ECG (ECG 90B).

ECG 90A

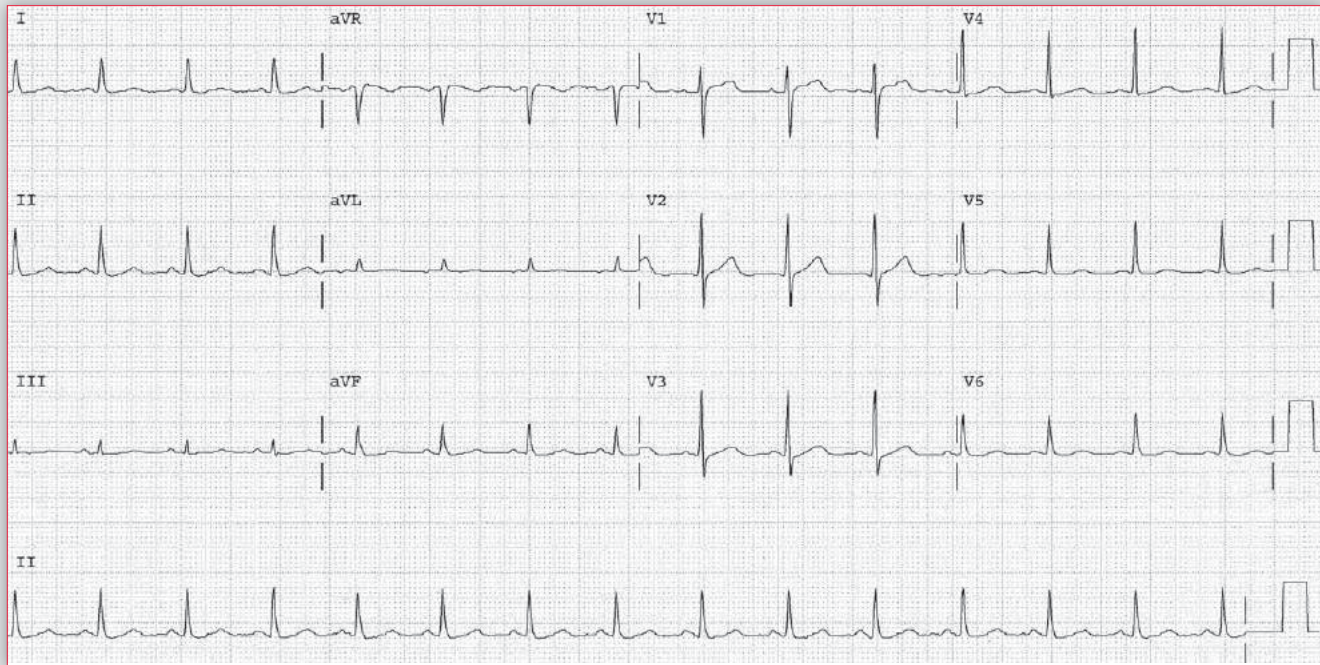


Practice Case 90

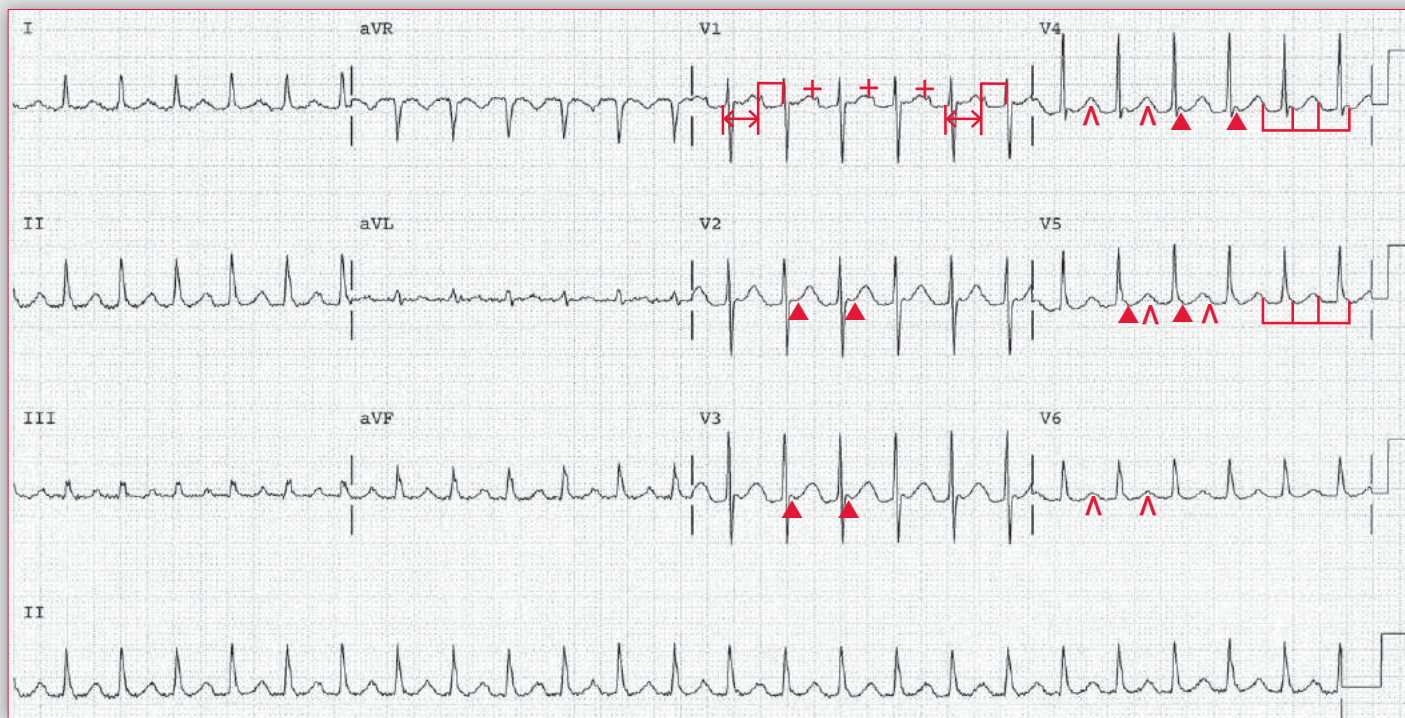
What is the rhythm abnormality?

Does this ECG help establish the etiology for the arrhythmia noted upon presentation?

ECG 90B



Podrid's Real-World ECGs



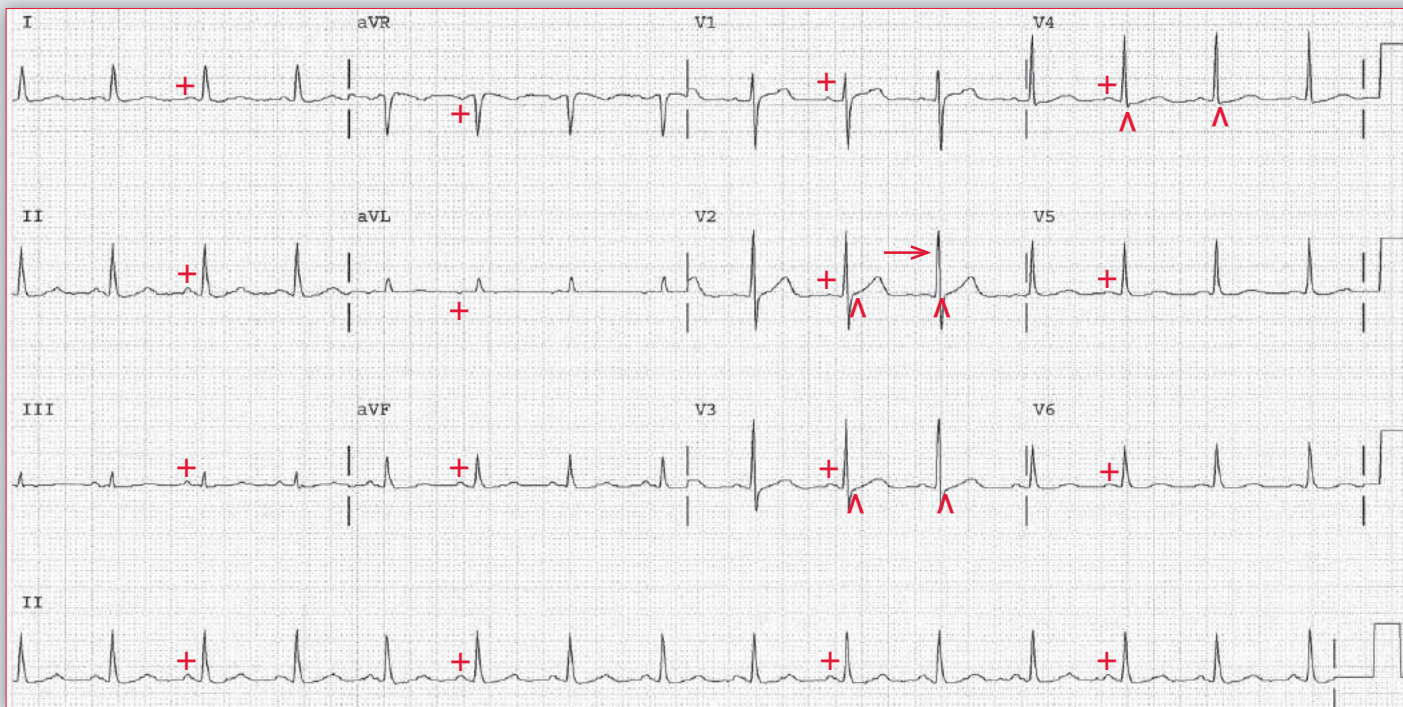
ECG 90A Analysis: Supraventricular tachycardia, long RP tachycardia, atrial flutter with 2:1 AV block

ECG 90A shows a regular rhythm at a rate of 148 bpm. The QRS complex duration is normal (0.08 sec) and there is a normal morphology. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/440 msec). Although P waves are not obvious, inspection of lead V1 reveals a P wave (+) superimposed on the downslope of the T wave. T waves should be smooth in upstroke and downstroke and any notches, bumps or other irregularities should be considered superimposed P waves. The PR interval is constant (0.18 sec). Having established the PR interval, it can be seen that there are notches on the downslope of the T waves (^) in leads V4–V6, which have the same relationship to the QRS complex, *ie*, a PR interval of 0.18 second. Hence, these are P waves. The P waves appear to be positive in these leads. However, as the P waves cannot be

seen in the limb leads, the etiology for the tachycardia is uncertain. The RP interval (↔) is 0.26 second, while the PR interval (□) is 0.18 sec. Hence this is a long RP tachycardia. Etiologies for a long RP tachycardia include sinus tachycardia, atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, an atypical or uncommon atrioventricular nodal reentrant tachycardia (impulse to ventricles via a fast pathway with retrograde conduction back to the atria via a slow), and atrioventricular reentrant tachycardia. The etiology remains uncertain. However, there is a second positive waveform seen after the QRS complex in leads V2–V4 (▲). The interval between this waveform and the P wave that is seen on the T wave is constant (⊐) at a regular rate of 300 bpm, which is diagnostic for atrial flutter with 2:1 AV block.

continues

Podrid's Real-World ECGs



ECG 90B Analysis: Normal sinus rhythm, counterclockwise rotation

ECG 90B is from the same patient as ECG 90A. There is a regular rhythm at a rate of 88 bpm. The QRS complex duration, morphology, and axis are identical to that seen in ECG 90A. However, there is a tall R wave in lead V2 (\rightarrow), consistent with early transition or counterclockwise rotation in the horizontal axis. This is established by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces are seen early in the precordial leads. The QT/QTc intervals are normal (360/440 msec). There is a P wave (+) seen before each QRS complex with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. It can be seen that the positive waveform after the QRS complex that was seen in leads V2–V4 on ECG 90A is not present (\wedge). Therefore, this was not part of the QRS

complex, but indeed represented a second atrial waveform, establishing the rhythm on ECG 90A as atrial flutter with 2:1 AV block.

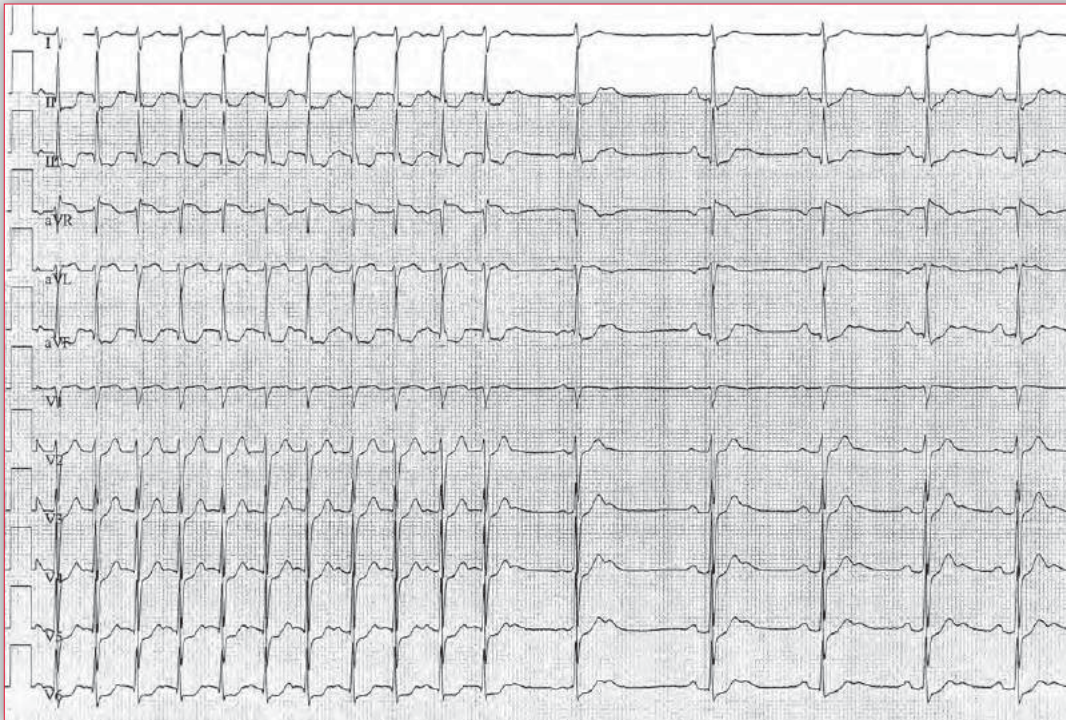
Typical atrial flutter is a result of a reentrant circuit within the right atrium, with an impulse that circulates around this circuit in a counterclockwise direction. Atrial flutter is an arrhythmia that has been observed to occur after pulmonary vein isolation for atrial fibrillation, as the scar formation after ablation can result in areas of reentry in the atria. However, this is not typical atrial flutter, as the reentrant circuit is within the left atrium. Left atrial flutter is often more difficult to treat as the circuit is in the left atrium and there is often diffuse scar formation resulting from prior left atrial ablation procedures. ■

Notes

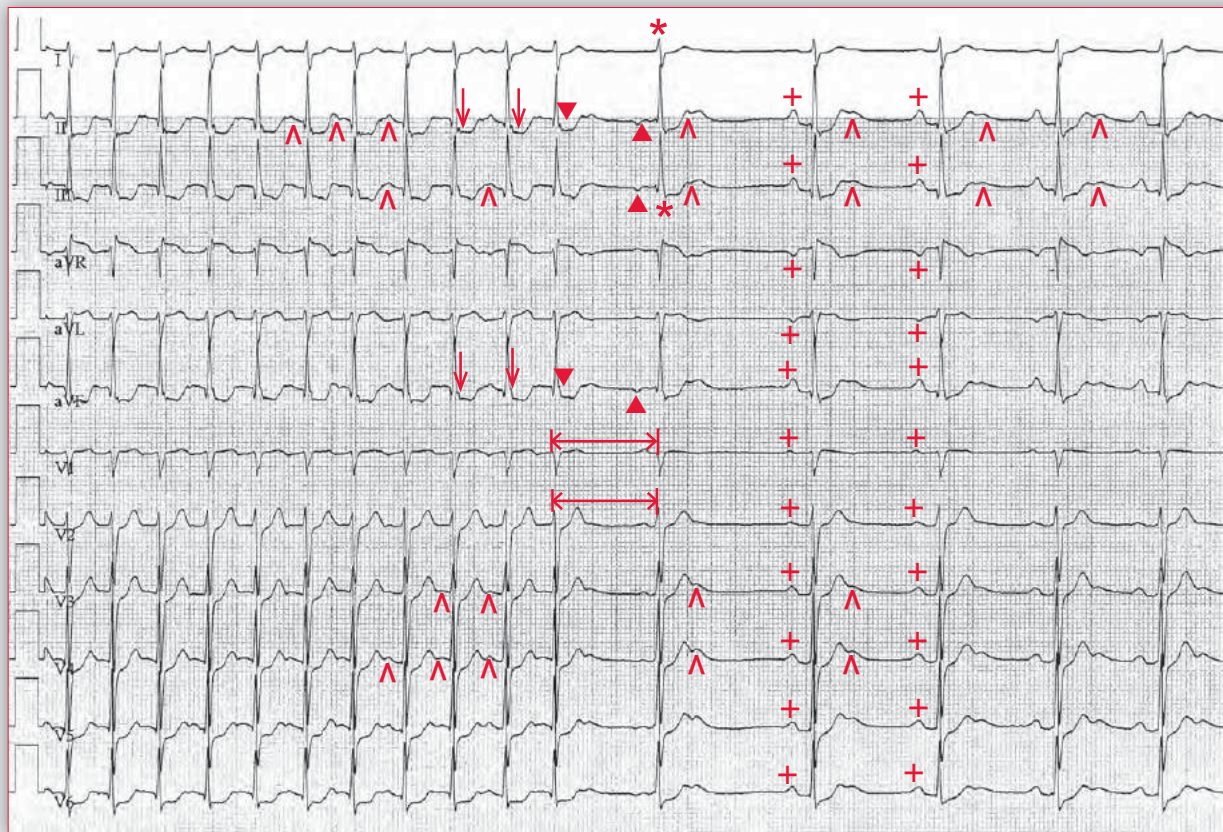
Practice Case 91

An otherwise healthy 21-year-old woman presents with new-onset palpitations after celebrating her birthday with friends at a bar. She denies a history of palpitations, syncope, or pre-syncope. The following ECG is obtained while the patient is symptomatic and during the performance of carotid sinus massage.

What is the most likely etiology of her arrhythmia?



Podrid's Real-World ECGs



ECG 91 Analysis: Narrow complex supraventricular tachycardia, atrioventricular nodal reentrant tachycardia, followed by sinus rhythm with first-degree AV block

ECG 91 shows that initially there is a regular rhythm at a rate of 140 bpm. The QRS complex has a normal duration (0.08 sec) and morphology. The axis is about 0° (QRS complex isoelectric in lead I and positive in lead aVF). The QT/QTc intervals are normal (280/430 bpm). There are no obvious P waves seen before or after any of the QRS complexes. Hence this might be termed a no-RP tachycardia, and the most common etiology for this type of narrow complex supraventricular tachycardia is an atrioventricular nodal reentrant tachycardia (AVNRT).

After the eleventh QRS complex, carotid sinus massage produces an abrupt termination of the tachycardia (↔) to a regular rhythm at a rate of 48 bpm that slowly accelerates to a rate of 66 bpm. There is a P wave before these QRS complexes (+) and except for the twelfth QRS complex (immediately following the pause or arrhythmia termination) the P wave is positive in leads I, II, aVF, and V4–V6. The PR interval is constant (0.22 sec). Hence this is a sinus rhythm with a first-degree AV block. However, the twelfth QRS complex (*), immediately after the pause (arrhythmia termination), has a P wave that is negative in leads II, and aVF (▲). This is, therefore, an atrial complex, probably an escape after the termination of the tachycardia.

Although there are no P waves seen before or after any of the initial QRS complexes, there are positive waveforms noted after the T waves in leads II, aVF, and V3–V4 (^). However, when comparing these QRS complexes and the T waves with those after the arrhythmia terminates,

it can be seen that these positive waveforms are also present (with the same relationship to the T waves) and are U waves, as can be seen during sinus rhythm. In contrast, there are subtle abnormalities of the terminal portion of the QRS complex, particularly evidenced in leads II and aVF (↓), which are not seen in the sinus complex. These are actually P waves superimposed on the terminal portion of the QRS complex. As indicated, this is termed a no-RP tachycardia. The most common etiology for this arrhythmia is an atrioventricular nodal reentrant tachycardia (AVNRT). Also noted is the fact that this superimposed P wave is seen with the last QRS complex before the rhythm terminates (▼). Hence the arrhythmia terminates with a nonconducted P wave and this is the mode of termination of arrhythmias that come from the AV node or junction, further supporting the fact that this is an AVNRT.

An AVNRT results from the presence of dual AV nodal pathways that are linked proximally and distally within the AV node. The slow pathway conducts slowly, but recovers quickly (short refractory period), while a fast pathway conducts more rapidly, but recovers slowly (long refractory period). If there is a premature atrial complex that occurs before the fast pathway has recovered, it will conduct to the ventricles via the slow pathway. If the fast pathway has recovered when the impulse reaches the distal connection between the two pathways, the impulse will conduct retrogradely back to the atria via the fast pathway, while conducting antegradely to the ventricles. If the impulse reaches the proximal connection of the two pathways when the slow

continues

Notes

pathway has recovered, it will reenter this pathway. Should this process continue, a reentrant arrhythmia (typical AVNRT) develops. As there is simultaneous activation of the atria (retrogradely via the fast pathway) and ventricles (antegradely via the slow pathway), no clear P wave is seen (P wave occurs during or at the end of the QRS complex), *ie*, a no-RP tachycardia.

Any intervention that alters the electrophysiologic properties of the AV nodal pathways has the potential to interrupt this circuit and terminate the arrhythmia. Pharmacologic therapy includes a β -blocker, calcium-channel blocker, digoxin, or adenosine. Vagal maneuvers (as was performed for this patient) can terminate an AVNRT by slowing conduction and prolonging refractoriness. As the slow pathway is

most vulnerable to these interventions, there is usually an antegrade AV block that occurs after there has been atrial activation. Hence this arrhythmia terminates with a nonconducted P wave as atrial activation has occurred prior to arrhythmia termination.

Vagal maneuvers can also terminate an atrioventricular reentrant tachycardia (AVRT) due to a circuit involving the AV node as well as an accessory pathway. The lack of pre-excitation (*ie*, a delta wave) in the patient's sinus beats favors the diagnosis of AVNRT over AVRT. However, there is still a possibility that the patient has a concealed bypass tract, which is only capable of retrograde conduction. Hence the ECG during sinus rhythm would look normal. However, an AVNRT is far more common than an AVRT due to a concealed bypass tract. ■

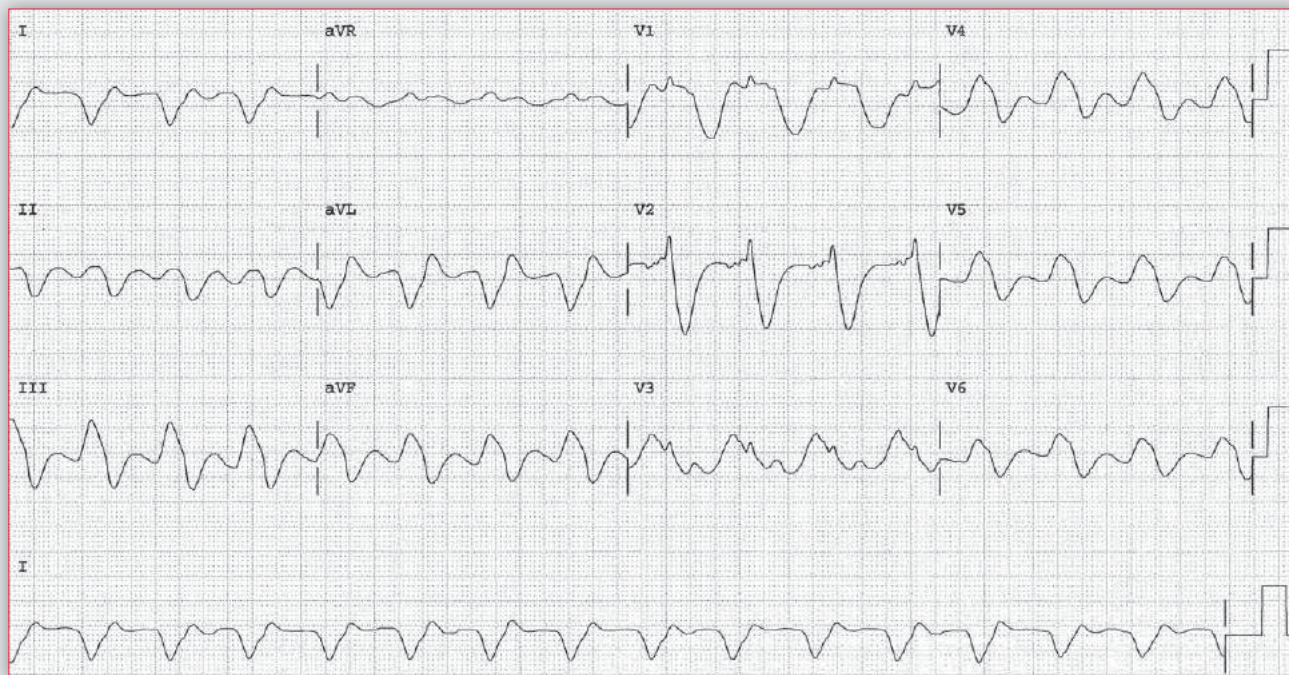
Practice Case 92

A 52-year-old female diabetic with systolic heart failure and Stage III chronic renal insufficiency is being treated with a β -blocker and ACE inhibitor. During a recent outpatient heart failure clinic appointment, therapy with an aldosterone antagonist (spironolactone) was initiated. One week later, the patient developed

nausea, vomiting, and weakness. In the emergency department, her blood pressure was noted to be 80/60 (previously it was normal). An ECG is obtained (ECG 92A).

Appropriate therapy was given and 2 hours later a new ECG is obtained (ECG 92B).

ECG 92A

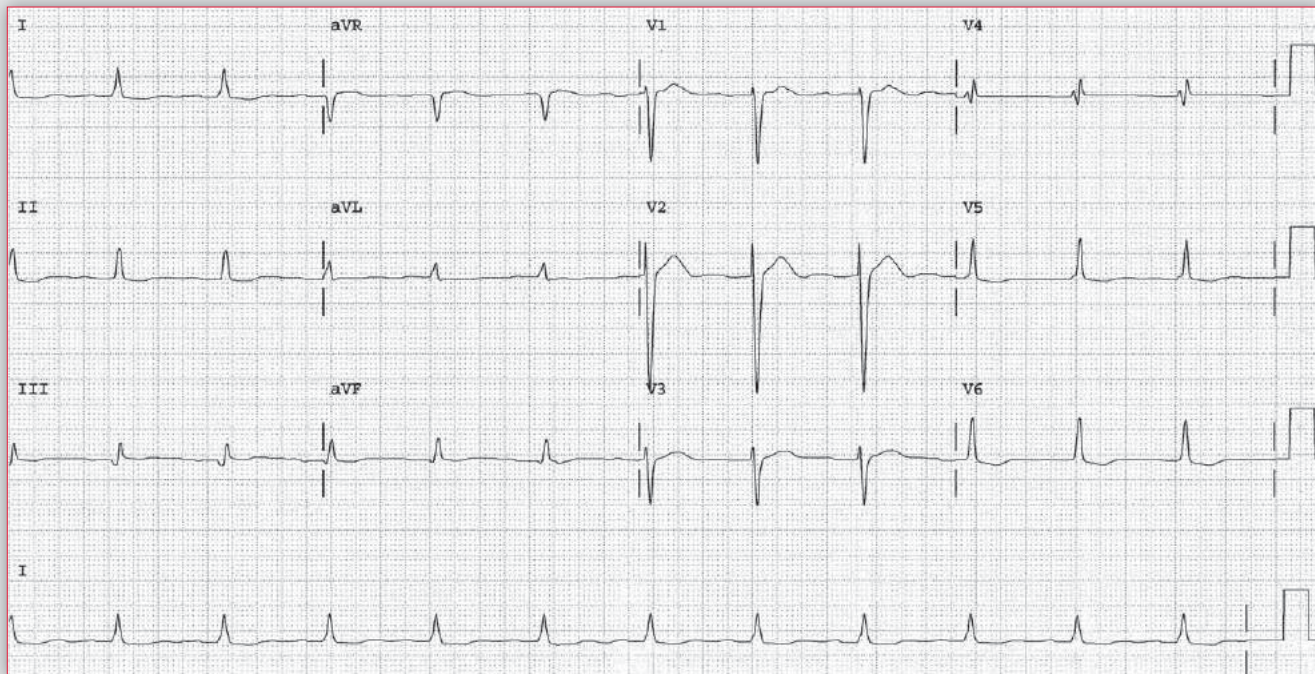


Practice Case 92

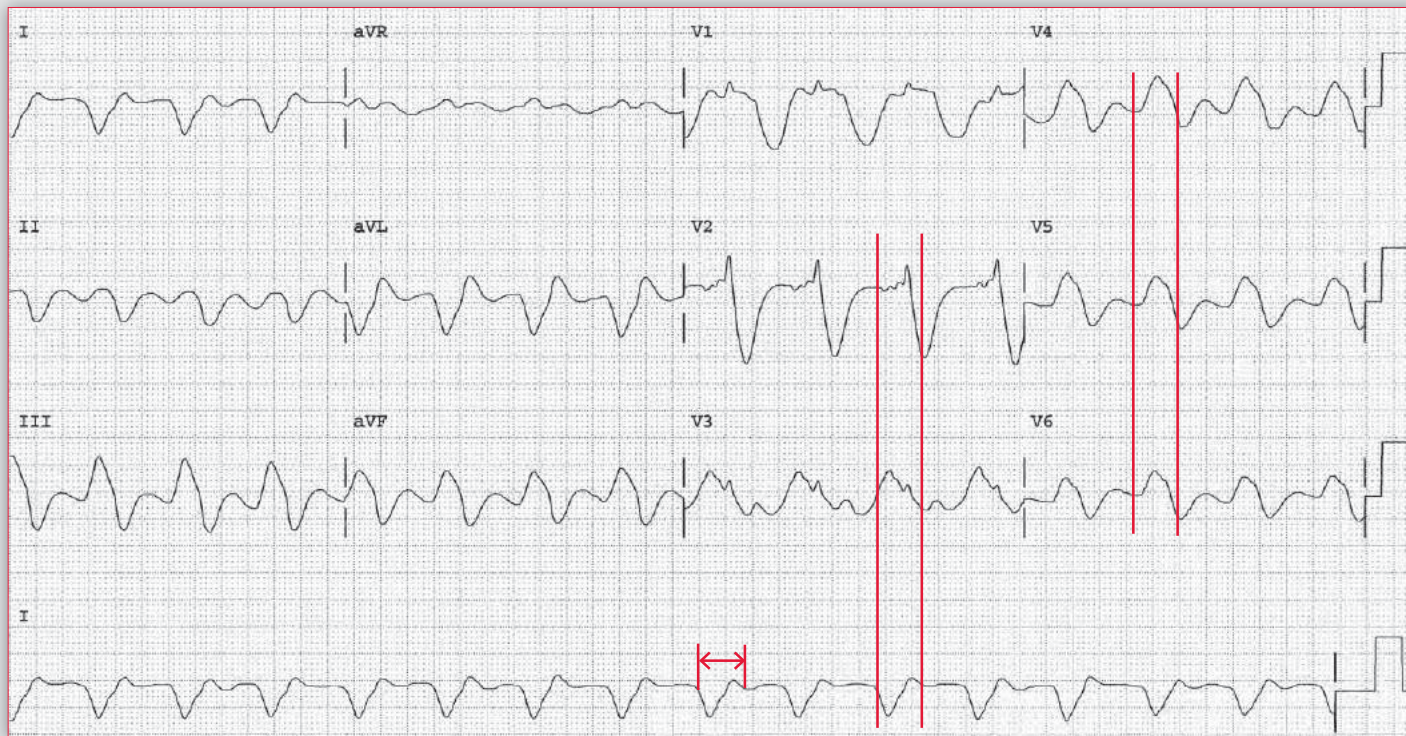
What does this show?

What therapy is indicated?

ECG 92B



Podrid's Real-World ECGs



ECG 92A Analysis: Regular rhythm, hyperkalemia

ECG 92A shows a regular rhythm at a rate of 98 bpm. There are no P waves seen before or after any of the QRS complexes. The waveforms noted in front of the QRS complex in lead V2 and after the QRS complex in lead V3 are not P waves but are actually part of the QRS complex, as established by measuring the maximal QRS complex duration (\parallel), for example in lead I as well as leads V4–V6. The QRS complex duration (\leftrightarrow) is extremely prolonged (0.36 sec). The only etiology for a QRS complex this wide (*ie*, ≥ 0.24 sec) is hyperkalemia, which causes diffuse slowing of the impulse conduction throughout the ventricular myocardium. This is due to the fact that with hyperkalemia, the resting membrane potential becomes less negative. The resting potential (-90 mV) is maintained by a balance between intracellular and extracellular potassium levels; intracellular potassium levels are higher than extracellular levels. An increase in extracellular levels will result in the membrane potential becoming less negative, approaching the membrane threshold potential of -60 mV (the potential at which a spontaneous action potential is generated as a result of a rapid influx of sodium ions, *ie*, phase 0). The closer the resting membrane potential is to the threshold potential, the slower is the influx of sodium ions and the slower is the upstroke velocity of phase 0. This upstroke velocity of phase 0 determines the rate of impulse conduction through the His-Purkinje system and ventricular myocardium. The slower the upstroke of phase 0, the slower is impulse conduction, and hence the wider is the QRS complex. Hence hyperkalemia slows the upstroke of phase 0,

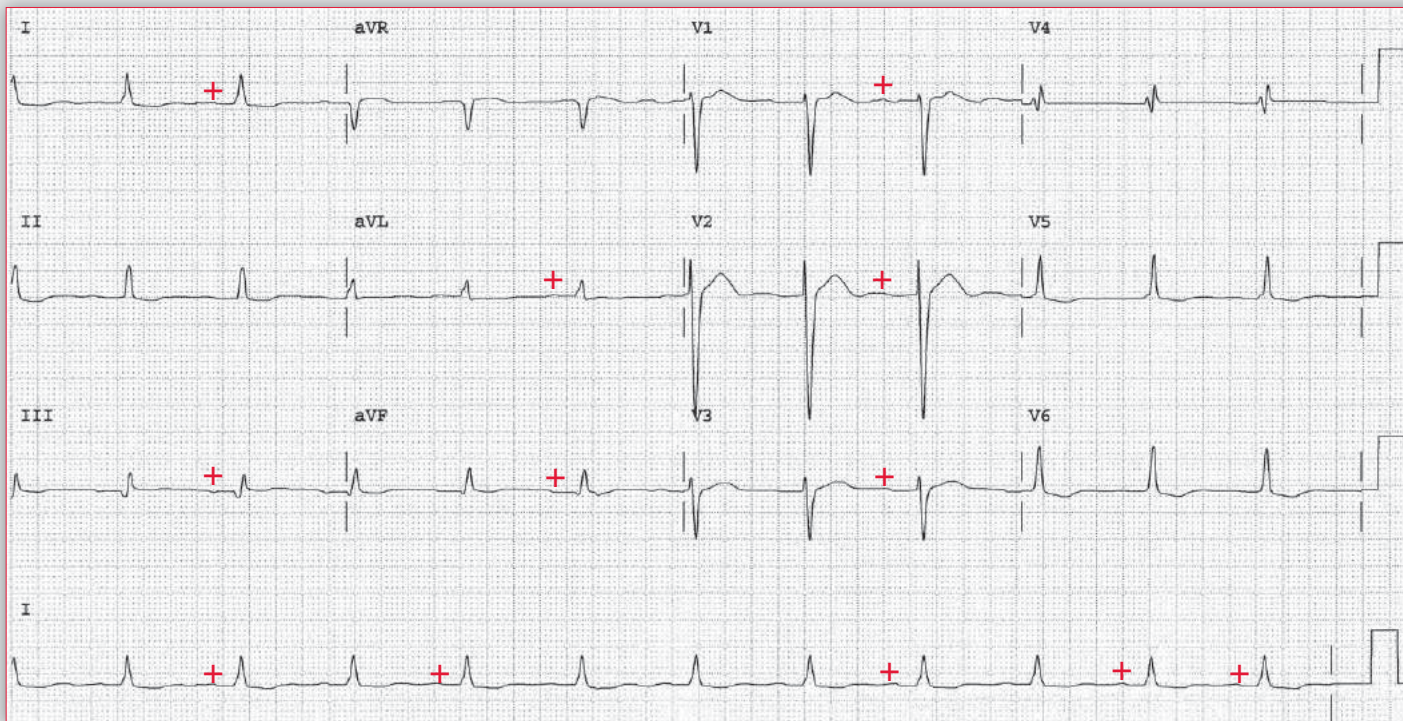
slows impulse conduction through the myocardium, and prolongs the QRS complex. Other etiologies for QRS complex widening include ventricular hypertrophy, any situation in which there is direct left ventricular myocardial activation (left or right bundle branch block, paced QRS complex, ventricular complex or Wolff-Parkinson-White), or when there is diffuse fibrosis of the ventricular myocardium such as a dilated cardiomyopathy. However, the only condition in which the QRS complex is ≥ 0.24 second is hyperkalemia. If the resting membrane potential reaches the membrane threshold potential, the myocardium is inexcitable and asystole occurs.

The etiology of the rhythm is not certain, and in the absence of P waves, it may be ventricular or junctional. However, it may be sinus as the atrial myocardium is more sensitive to hyperkalemia than the ventricular myocardium and atrial asystole may occur even before QRS complex widening. In this situation, there is sinus node activity but no activation of the atrial myocardium and hence no P wave. This is termed a sinoventricular rhythm.

Although aldosterone antagonists have demonstrated a mortality benefit in patients with systolic heart failure, they must be used with caution in patients with renal insufficiency, particularly when also receiving an ACE inhibitor or an angiotensin II receptor blocker (ARB) due to the potential complication of hyperkalemia.

continues

Podrid's Real-World ECGs



ECG 92B Analysis: Normal sinus rhythm, first-degree AV block, diffuse nonspecific T-wave abnormalities

ECG 92B is of the patient with ECG 92A and was obtained several hours after treatment. There is a regular rhythm at a rate of 70 bpm. Although not well seen, there is a P wave before each QRS complex (+), most obvious in leads I, III, aVL, aVF, and V1–V3. The P wave appears to be positive in leads I and aVF, and this is likely a normal sinus rhythm. The PR interval is constant (0.22 sec), with a first-degree AV block. The QRS complex duration is normal (0.10 sec) and there is a normal morphology. The axis is normal between 0° and $+90^{\circ}$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/410 msec). The T waves are flat in many of the leads, which is a nonspecific abnormality.

The treatment of hyperkalemia is to acutely lower the serum potassium level using insulin (and glucose to prevent hypoglycemia), which forces potassium into the cells. Calcium is also recommended to increase impulse conduction velocity, although the mechanism by which this happens is not established. In addition, therapy to lower the total body potassium level is begun, *ie*, Kayexelate. ■

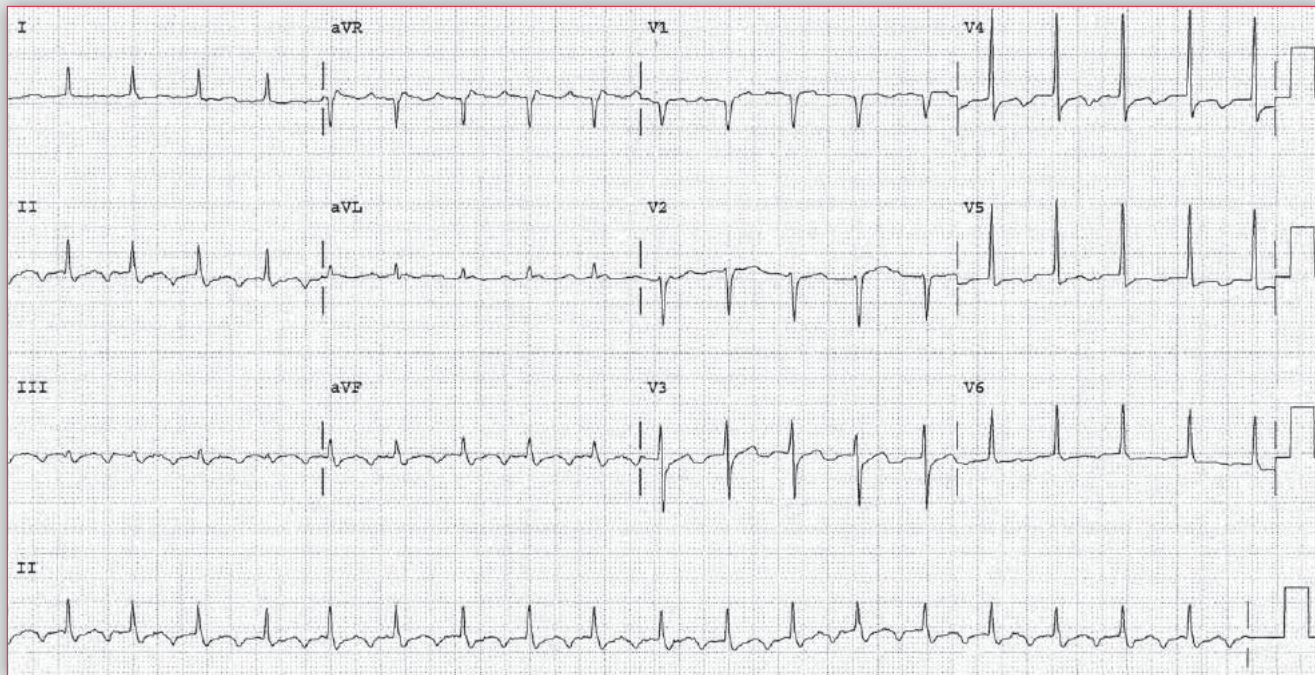
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Practice Case 93

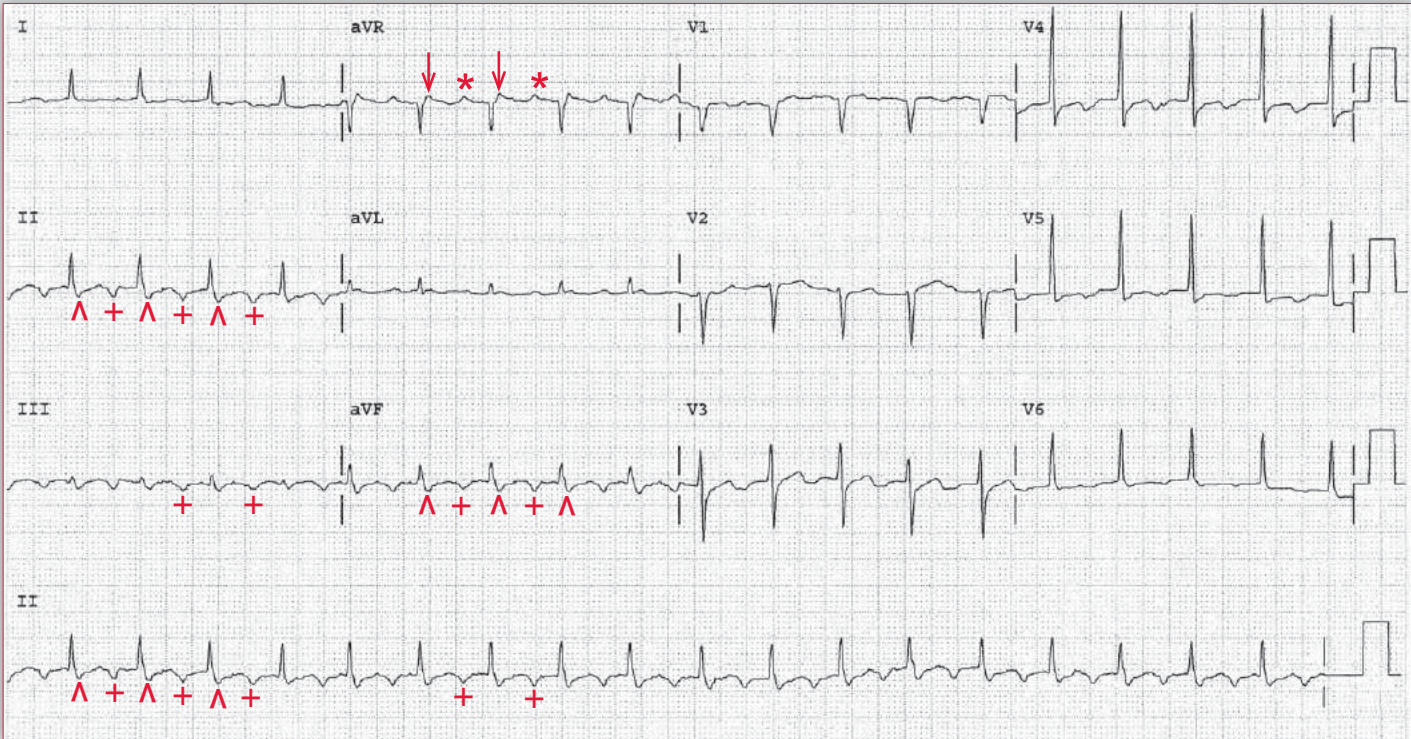
You are consulted on an 80-year-old patient with diabetes and hypertension who has the following ECG. You are told that the patient has had similar ECGs in the past.

What is the rhythm abnormality?

What are your recommendations with regard to chronic anticoagulation?



Podrid's Real-World ECGs



ECG 93 Analysis: Atrial flutter with 2:1 AV block

ECG 93 shows a regular rhythm at a rate of 120 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/400 msec). There is negative atrial activity (+) seen in leads II, III, and aVF while the atrial activity is positive in leads aVR and aVL (*). This is a long RP tachycardia (RP = 0.30 sec, PR = 0.20 sec). Etiologies include sinus tachycardia (eliminated given the negative P wave in II and aVF), atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atypical atrioventricular nodal reentrant tachycardia (known as fast-slow, with antegrade activation of the ventricles the fast pathway and retrograde atrial activation via the slow pathway), or atrioventricular reentrant tachycardia. However, a second atrial waveform can be seen immediately after the QRS complex in lead aVR (\downarrow), corresponding to what looks like an S wave in leads II, III, and aVF (\wedge). This second atrial waveform occurs at a regular interval (rate 240 bpm). At this atrial rate, the rhythm is either an atrial tachycardia that is slightly faster than usual or an atrial flutter with 2:1 AV block that is slightly slower than usual. It appears that there is continuous undulation of the

baseline between each atrial waveform (without an isoelectric baseline between each atrial waveform); hence this is most likely atrial flutter with 2:1 AV block.

Although the actual risk of a thromboembolic event with atrial flutter is uncertain, the current guidelines recommend the same considerations in regard to anticoagulation for atrial flutter as for atrial fibrillation. Based on the CHADS₂ (or CHA₂DS₂-VASc) score, risk factors for thromboembolic complications include CHF (1 point), Hypertension (1 point), Age > 75 (1 point), Diabetes (1 point), and Stroke (or any thromboembolic) history (2 points). A CHADS₂ score of ≥ 2 warrants long-term anticoagulation with warfarin, whereas high-dose aspirin is sufficient for a score of 0. A patient with a score of 1 can be treated with either aspirin or warfarin. This patient has a CHADS₂ score of 3 (age, diabetes, hypertension) and requires long-term anticoagulation with warfarin, as long as there is no significant bleeding history or risk for bleeding. Direct thrombin inhibitors are now approved for long-term anticoagulation in patients with atrial fibrillation. These oral anticoagulants do not require INR monitoring. ■

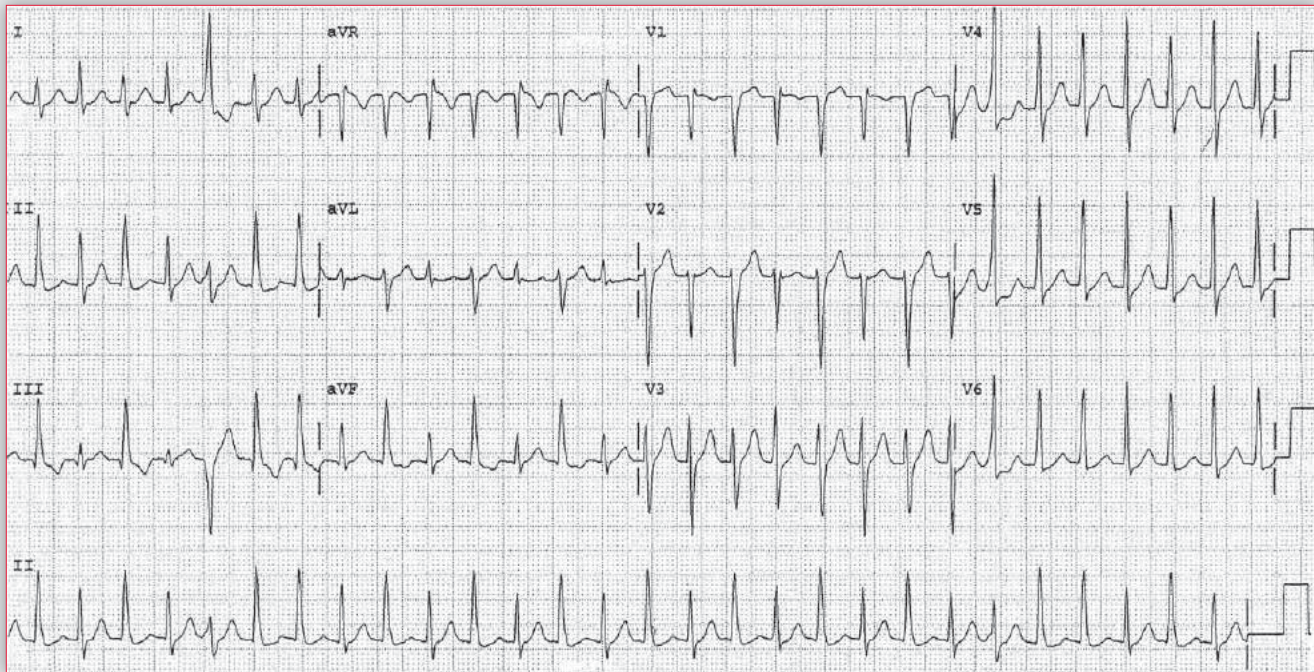
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Practice Case 94

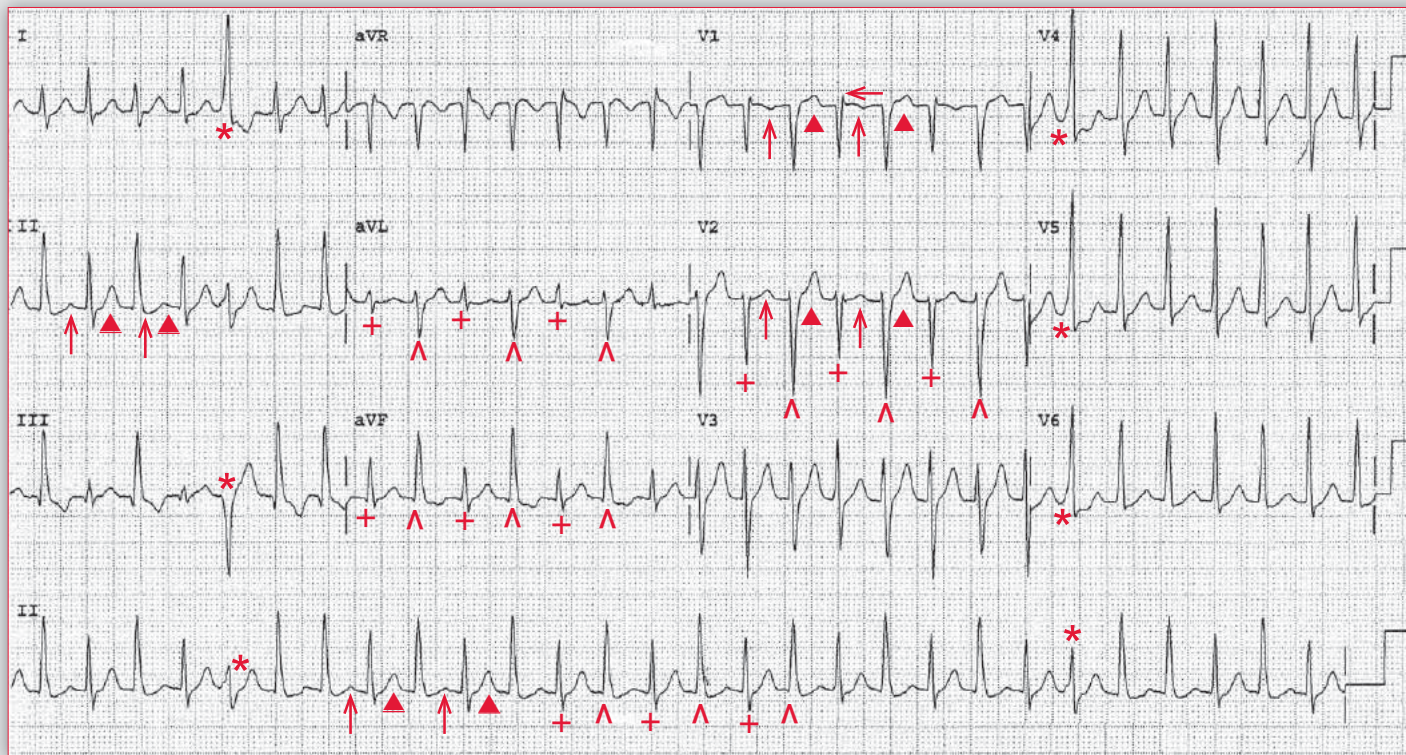
An otherwise healthy 24-year-old woman presents with palpitations during the third trimester of pregnancy. Her blood pressure is 120/80.

What is the most likely rhythm abnormality?

What is the mechanism of the beat-to-beat variation in the QRS complexes?



Podrid's Real-World ECGs



ECG 94 Analysis: Narrow complex supraventricular tachycardia, no-RP tachycardia, typical AVNRT, electrical (QRS) and T-wave alternans, premature ventricular complexes

ECG 94 shows a regular rhythm at a rate of 160 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (240/390 msec).

There are two QRS complexes (fifth and twenty-third) (*) that are premature, have a longer duration (0.12 sec), and have a different morphology. These are premature ventricular complexes. The premature ventricular complexes do not affect the underlying RR intervals or heart rate.

Although all the other QRS complexes have the same duration and a similar morphology, there are beat-to-beat changes in QRS amplitude (\uparrow, \wedge), which is known as QRS or electrical alternans. In lead V1 there is not only a beat-to-beat change in QRS amplitude, there is also an R' (\leftarrow) that occurs in every other beat. This is a slight right ventricular conduction delay that is also occurring with every other complex. In addition, there are beat-to-beat changes in T-wave amplitude, most obvious in leads II, III, aVF, and V1–V2 (\blacktriangle, \uparrow), known as T-wave alternans.

There are no obvious P waves seen before or after any of the QRS complexes. This is therefore a no-RP tachycardia and the most likely cause

is a common or typical atrioventricular nodal reentrant tachycardia, known as slow-fast. In this situation, there are dual AV nodal pathways, *ie*, a slow pathway that conducts slowly but repolarizes quickly and a fast pathway that conducts rapidly but repolarizes slowly. The antegrade conduction to the ventricles is via the slow pathway while retrograde activation of the atria is via the fast pathway, resulting in simultaneous activation of the atria and ventricles, and hence no obvious P waves.

Electrical alternans occurs in various conditions, including cardiac tamponade, a severe dilated cardiomyopathy, decompensated heart failure, or an acute myocardial infarction. It may also be seen in association with any rapid, regular supraventricular tachycardia, including atrial flutter, atrial tachycardia, atrioventricular nodal reentrant tachycardia, or atrioventricular reentrant tachycardia. In rapid supraventricular tachycardias, myocardial infarction, decompensated heart failure, and a severe dilated cardiomyopathy, the electrical alternans is due to beat-to-beat changes in calcium fluxes. In contrast, electrical alternans seen with a large pericardial effusion or tamponade is due to the beat-to-beat swinging motion of the heart. Since there is an anatomic shift in the heart with tamponade, there may also be P-wave alternans, reflecting changes in atrial impulse direction. P-wave alternans is not seen in the other conditions associated with QRS and T-wave alternans. ■

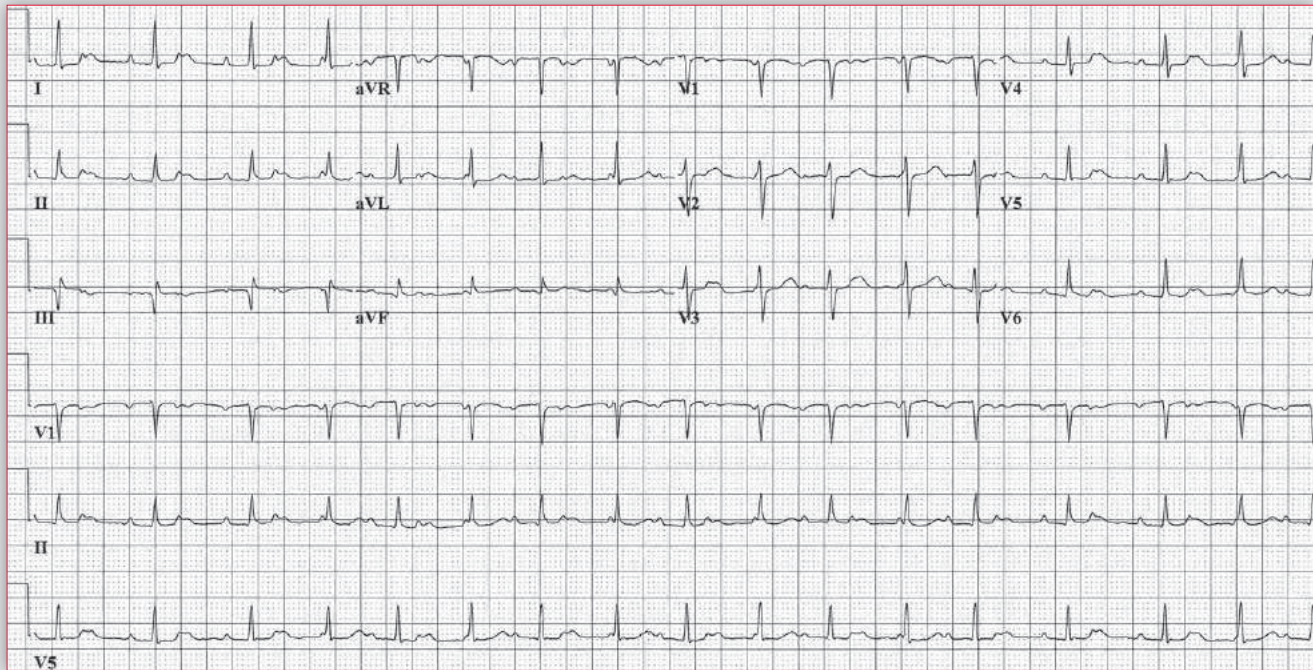
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Practice Case 95

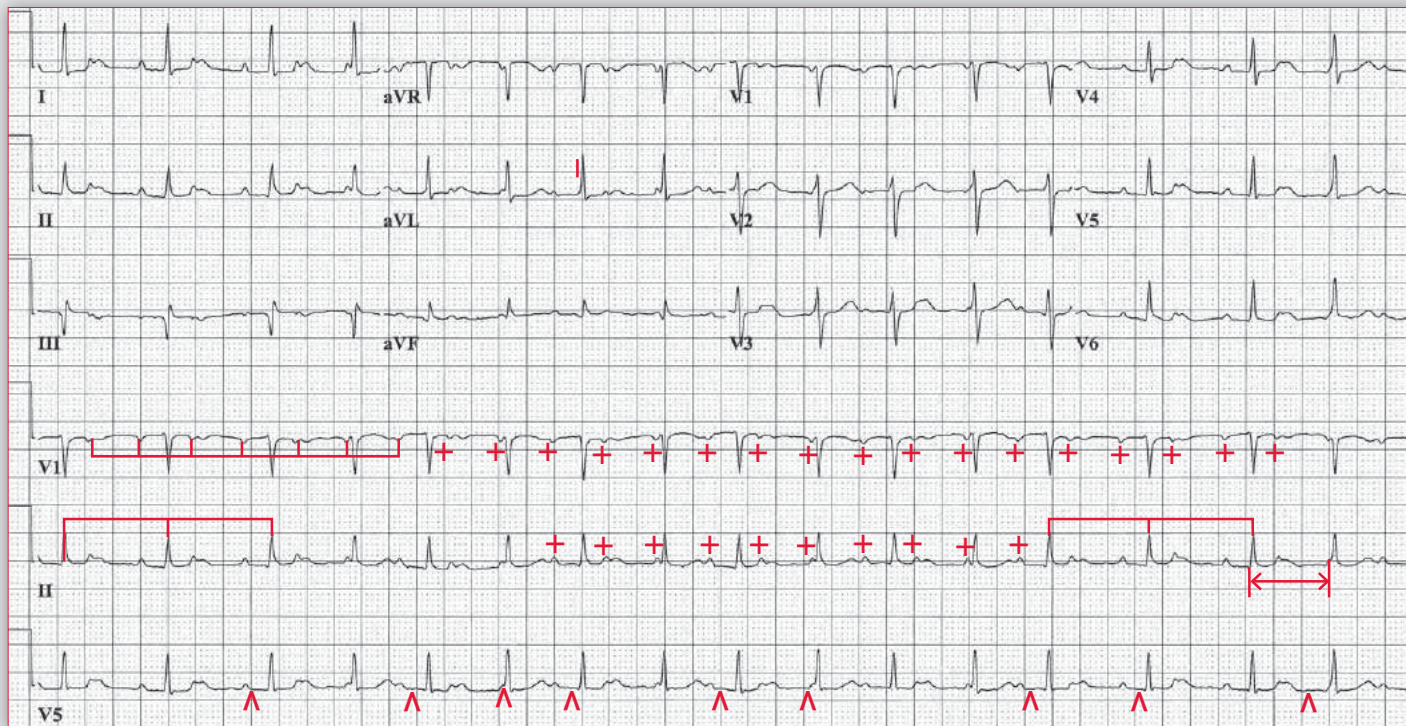
The following ECG is taken from an asymptomatic patient who is being monitored on telemetry after an appendectomy. The nurses noted that his heart rate has become slightly faster, but also irregular. The primary team is worried that the patient needs a pacemaker due to high-grade heart block.

What is the rhythm abnormality?

Does this patient need a pacemaker?



Podrid's Real-World ECGs



ECG 95 Analysis: Atrial tachycardia with variable AV block, antegrade-concealed conduction

ECG 95 shows that although there is a relatively regular rate of 102 bpm, there are subtle differences in some of the RR intervals, *ie*, some are slightly longer (\leftrightarrow) than others (\square). However, all of the longer RR intervals are the same, and the shorter intervals are also the same. Hence this is a regularly irregular rhythm. The QRS complexes have a normal duration (0.08 sec) and axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/440 msec).

There are distinct P waves (+) seen at a constant rate of 150 bpm (\sqcup). The P waves are upright in leads I, II, aVF and V4–V6. They appear to be dissociated from the QRS complex, *ie*, there is variability of the PR intervals (\wedge). However, the QRS complex (RR) intervals are not regular; hence this is not a junctional escape rhythm. The irregularity of the RR intervals means that the ventricles are responding in an irregular fashion to the atrial impulses, reflecting variable conduction through the AV node. This is the result of concealed antegrade

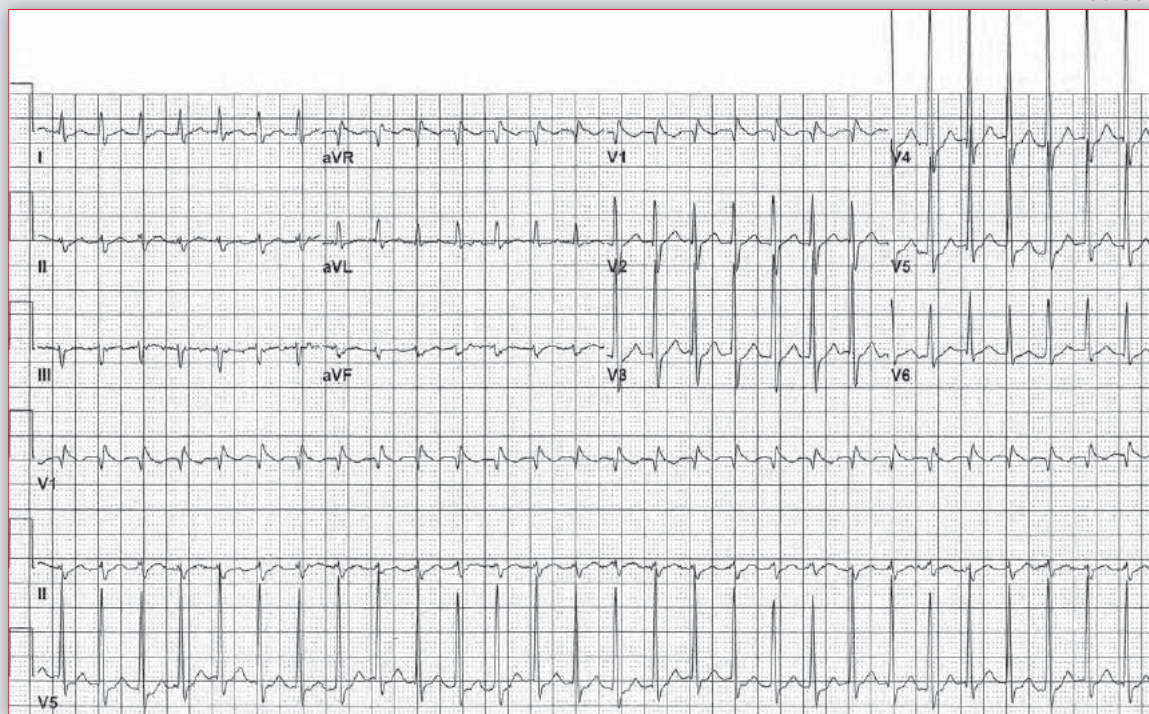
conduction, which is often seen with atrial tachycardia as well as atrial flutter. At this rapid atrial rate (and in the absence of sympathetic stimulation), some of the atrial impulses penetrate the AV node completely while others are blocked. However, some atrial impulses partially penetrate the AV node (*ie*, concealed within the AV node), partially depolarizing it and slowing the rate of AV conduction of the next atrial impulse.

Although the P waves have a morphology suggesting a sinus tachycardia, this irregular pattern of changing PR intervals and RR intervals is not seen with sinus tachycardia. AV nodal conduction abnormalities with sinus rhythm would present as Wenckebach, or a fixed degree of block (*ie*, 2:1, 3:1). If complete heart block were present, there would be an escape rhythm that would be slower than the atrial rate, but with a stable rate or RR interval. This is not the case with this rhythm. Hence this is an atrial tachycardia with variable AV block and the patient does not have an indication for a pacemaker. ■

Practice Case 96

A 42-year-old woman with a recent history of viral myocarditis presents with palpitations and dizziness. An ECG is obtained while the patient is symptomatic (ECG 96A). The ECG is compared to a recent ECG obtained during the acute myocarditis (ECG 96B).

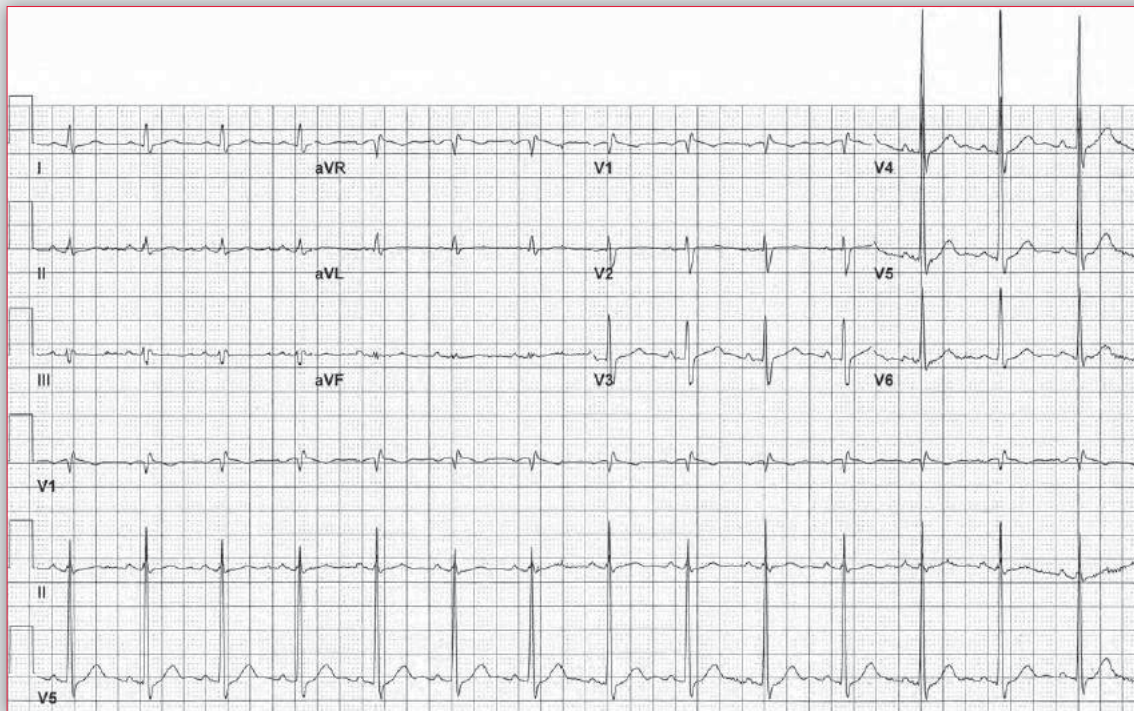
ECG 96A



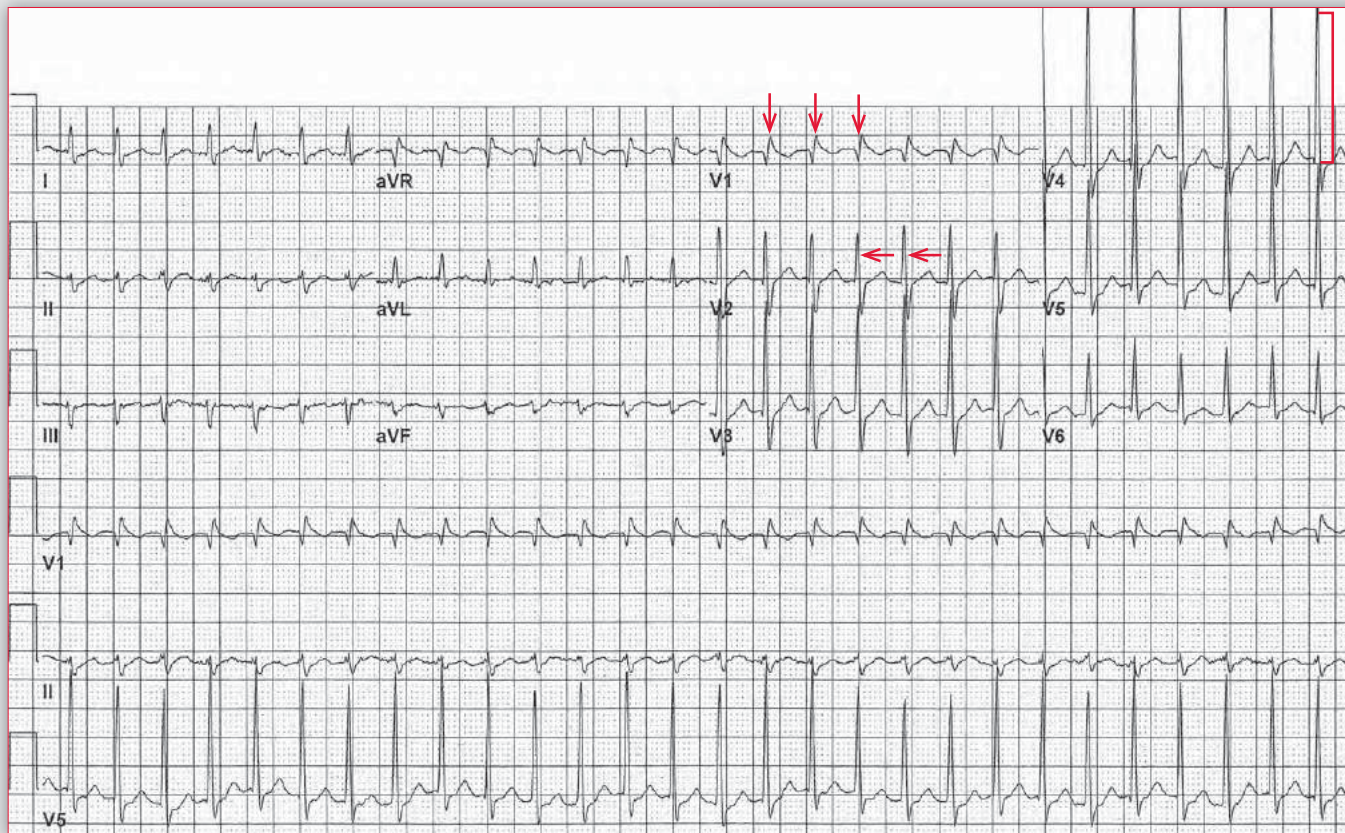
Is there any evidence for atrial activity?

What is the rhythm abnormality?

ECG 96B



Podrid's Real-World ECGs



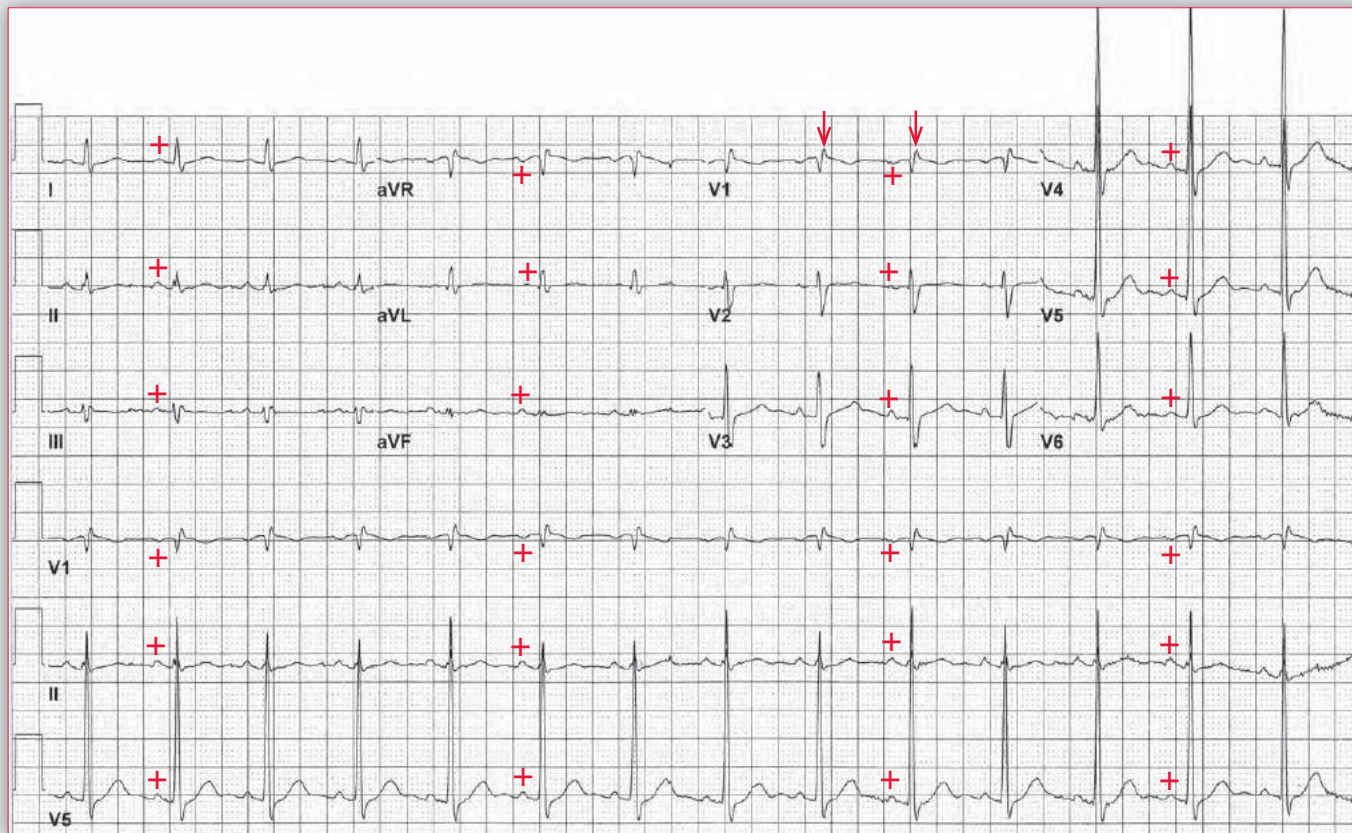
ECG 96A Analysis: Supraventricular tachycardia, no RP tachycardia, AVNRT, left axis anterior fascicular block, left ventricular hypertrophy, counterclockwise rotation

ECG 96A shows a regular rhythm at a rate of 170 bpm. The QRS complex duration is normal (0.10 sec), and there is an extremely leftward axis between -30° and -90° (QRS complex positive in lead I and negative in leads II and aVF with an rS complex). This is termed a left anterior fascicular block. There is also left ventricular hypertrophy as indicated by an R wave in lead V4 = 28 mm (\uparrow), which fulfills one of the criteria for left ventricular hypertrophy (*ie*, R or S wave > 25 mm in any one precordial lead). In addition, there is a tall R wave in lead V2 (\leftarrow), a result of early transition or counterclockwise rotation in the horizontal plane. This is determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the electrical forces from the left ventricle are seen more anteriorly, and become obvious in the early precordial leads. The QT/QTc intervals are normal (260/440 msec).

There are no obvious P waves seen before or after any QRS complex. However, there is an R' in lead V1 (\downarrow) which may be either the normal QRS complex morphology or be the result of a retrograde P wave (superimposed on the end of the QRS complex). This can be established by comparison with an ECG in normal sinus rhythm. Regardless of whether or not this is a superimposed P wave, there is no obvious P wave seen and hence this might be termed a no-RP tachycardia. The most likely etiology is a typical atrioventricular nodal reentrant tachycardia.

continues

Podrid's Real-World ECGs



ECG 96B Analysis: Normal sinus rhythm, left ventricular hypertrophy

ECG 96B is from the same patient as ECG 96A. There is a regular rhythm at a rate of 86 bpm. The QRS complex duration, morphology, and voltage are the same as seen in ECG 96A, although early transition is not present. There is an R' seen in lead V1 (↓), which is the same as noted in ECG 96A; hence this not a superimposed P wave but represents the native QRS complex morphology. The QT/QTc intervals are the same as in ECG 96A.

There is a P wave seen before each QRS complex (+) with a constant PR interval (0.16). The P wave is positive in leads I, II, aVF, and V4–V6; hence this is a normal sinus rhythm. By comparing the two ECGs, it is clear that there is no evidence for atrial activity on ECG 96A, further supporting the diagnosis of a common or typical atrioventricular nodal reentrant tachycardia with QRS complexes that are identical to those seen in ECG 96A. ■

Practice Case 97

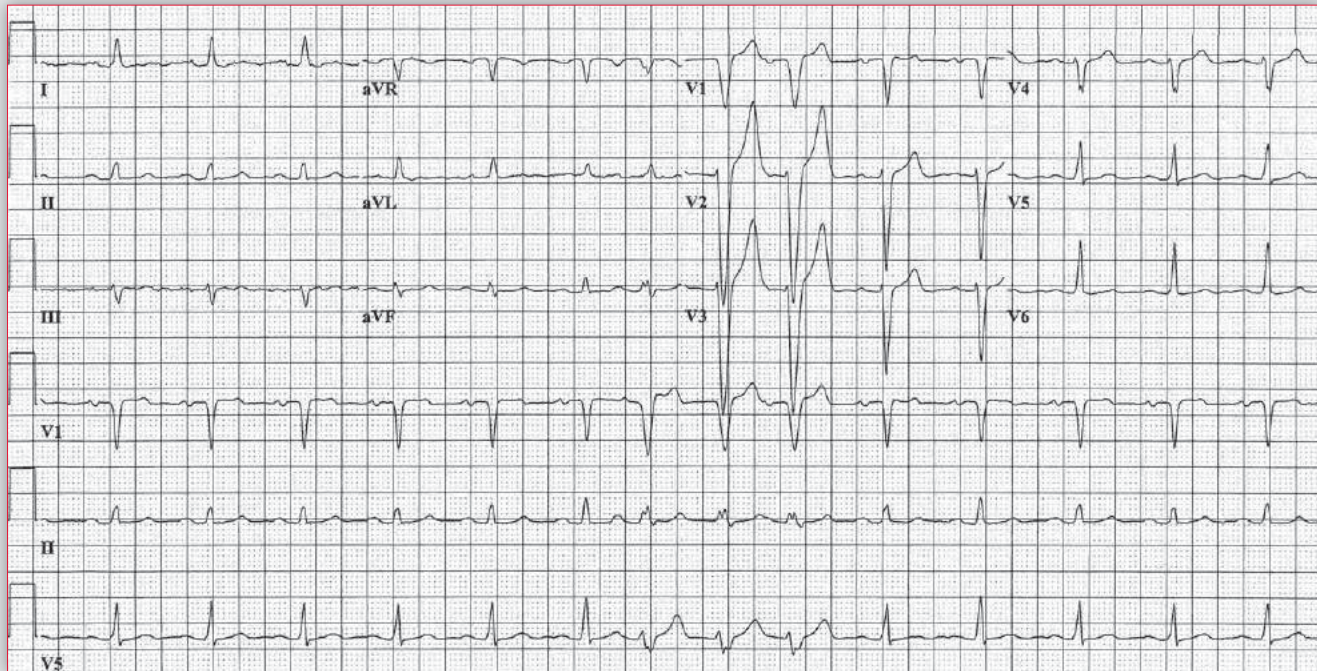
A 44-year-old female presents with complaints of a “racing heart.” She has noted this symptom on and off for the past month. She states that on occasion, she has noted a panicked feeling associated with sweating.

On review, she notes an upper respiratory

infection one week prior and currently has a “sore throat.” She does not carry any active diagnoses and does not take medications regularly.

An ECG is obtained (ECG 97A). During the visit, she notes sudden onset of her symptoms and a repeat ECG is obtained (ECG 97B).

ECG 97A



Practice Case 97

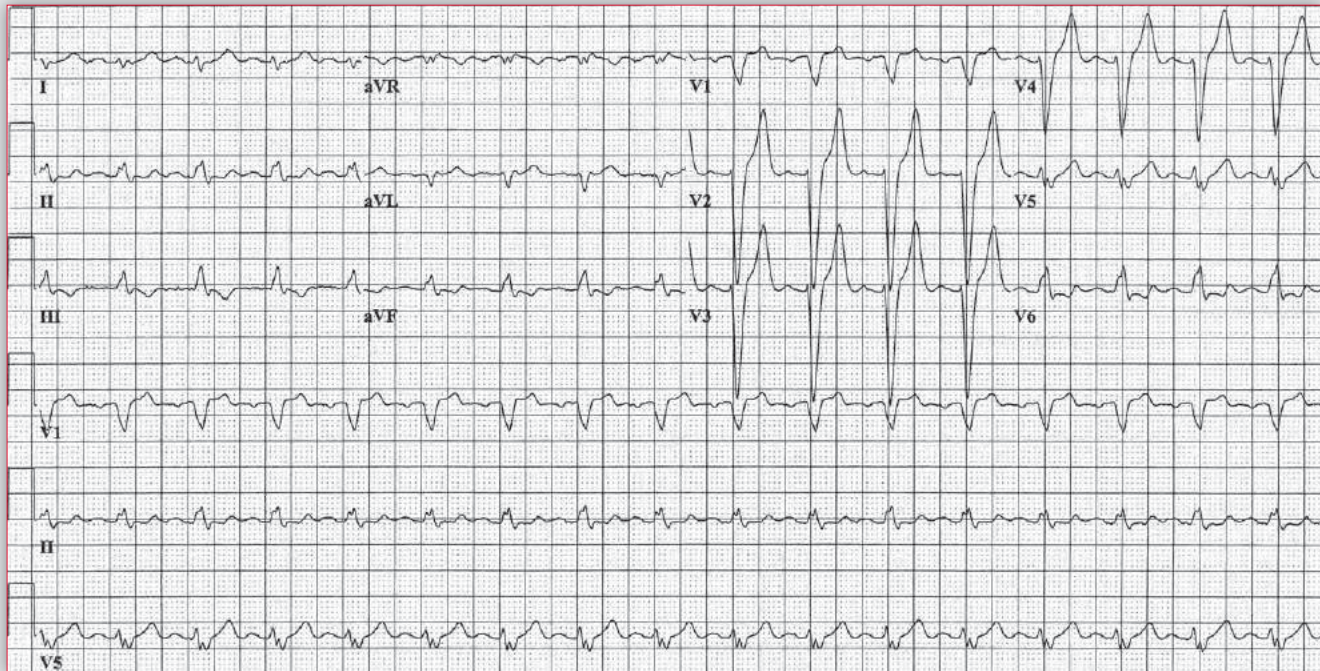
What diagnosis is suggested for ECG 97A?

What diagnosis is confirmed by ECG 97B?

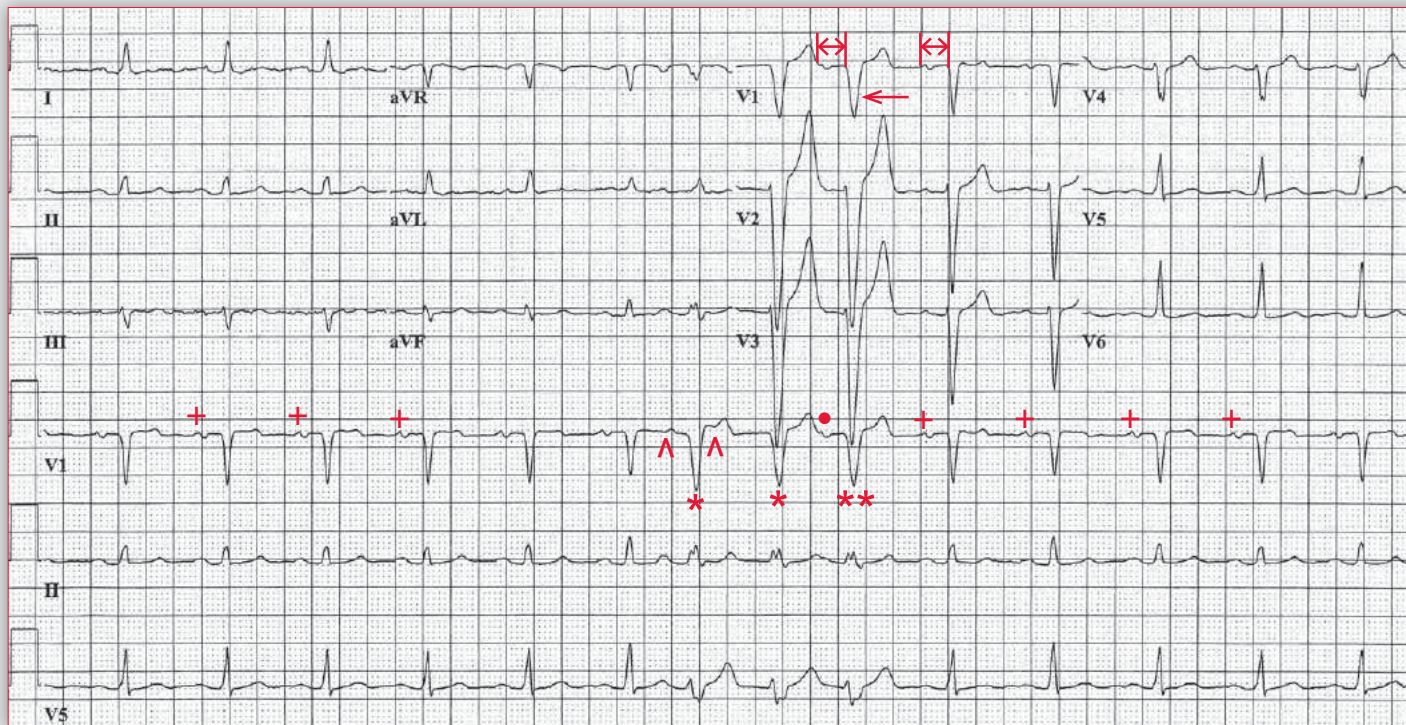
What clinical entity is suggested by the history?

What further testing should be done?

ECG 97B



Podrid's Real-World ECGs



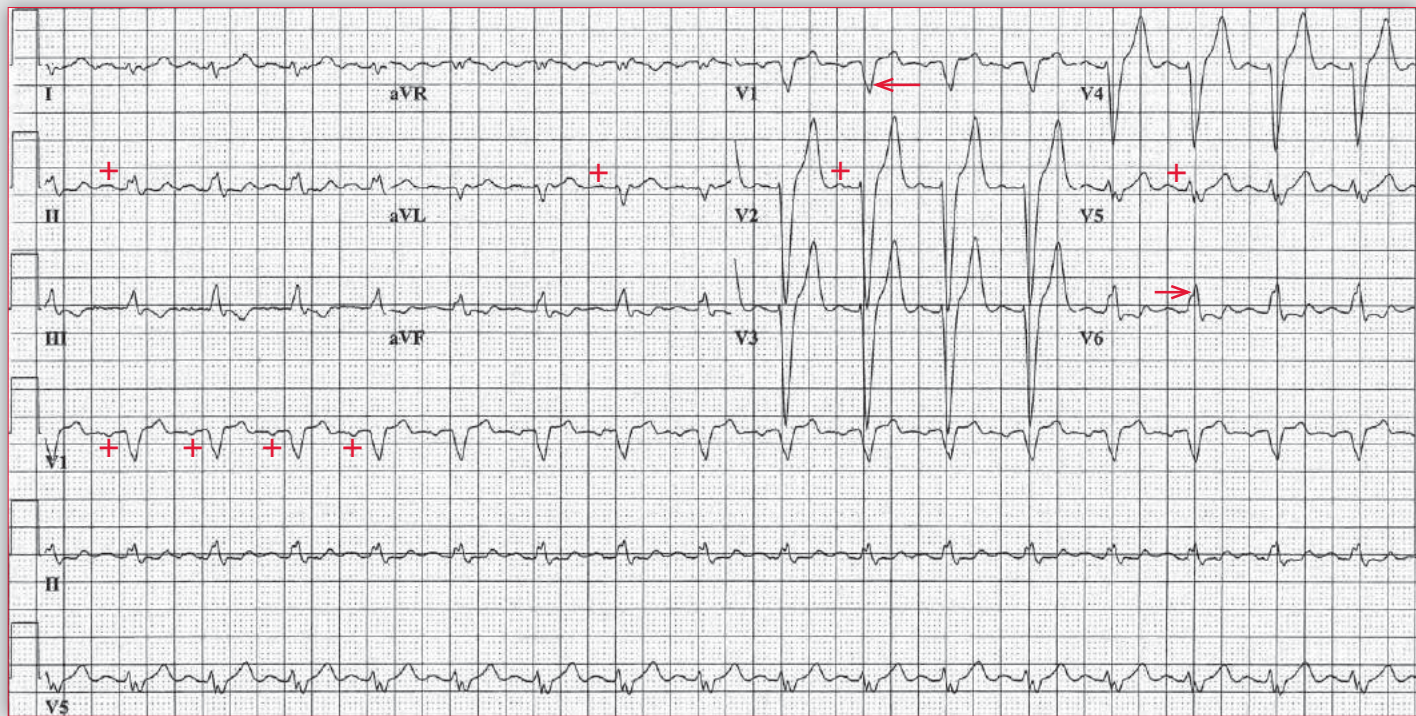
ECG 97A Analysis: Normal sinus rhythm, premature atrial complexes with rate-related left bundle branch morphology

ECG 97A shows a regular rhythm at a rate of 88 bpm, although the seventh, eighth, and ninth QRS complexes (*,**) are irregular in occurrence, with shorter RR intervals. There is a P wave (+) in front of each of the regular QRS complexes with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. These are sinus complexes.

The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/410 msec). The QRS complexes in the limb leads have low voltage (*ie*, < 5 mm or little boxes in each lead). They otherwise have a normal morphology.

The seventh and eighth QRS complexes (*) are premature. There is a P wave (^) (of a different morphology compared to the sinus P wave) before both of them, but the PR interval is not constant and is different from the PR interval of the sinus complexes. Therefore, these are premature atrial complexes. The QRS complexes are wide (0.14 sec) and have a left bundle branch block morphology (broad QS complex in lead V1 [←] and broad R wave in lead II. Hence these are premature atrial complexes with a rate-related left bundle branch block. The ninth QRS complex (**) has a P wave (●) before it with the same morphology as the sinus P waves. In addition, the PR interval is the same as the sinus complexes (↔). Hence this is a sinus beat with a short RR interval (between it and the eighth complex). Therefore, it also demonstrates a rate-related left bundle branch block.

continues



ECG 97B Analysis: Sinus tachycardia with a rate-related left bundle branch block

ECG 97B is from the same patient as ECG 97A. There is a regular rhythm at a rate of 100 bpm. There is a P wave (+) before each QRS complex and it has the same morphology as seen in ECG 97A. This is a sinus tachycardia. In addition, the PR interval is also the same (0.20 sec) as in ECG 97A. The QRS complexes duration is increased (0.14 sec), and there is a left bundle branch morphology (broad QS complex in lead I [←] and broad R wave in lead V6 [→]) that is identical to the morphology of the premature atrial complexes in ECG 97A. As the sinus rate is faster on this ECG, this is a rate-related left bundle

branch block. This confirms that the premature complexes seen in ECG 97A are atrial with a left bundle branch block aberration.

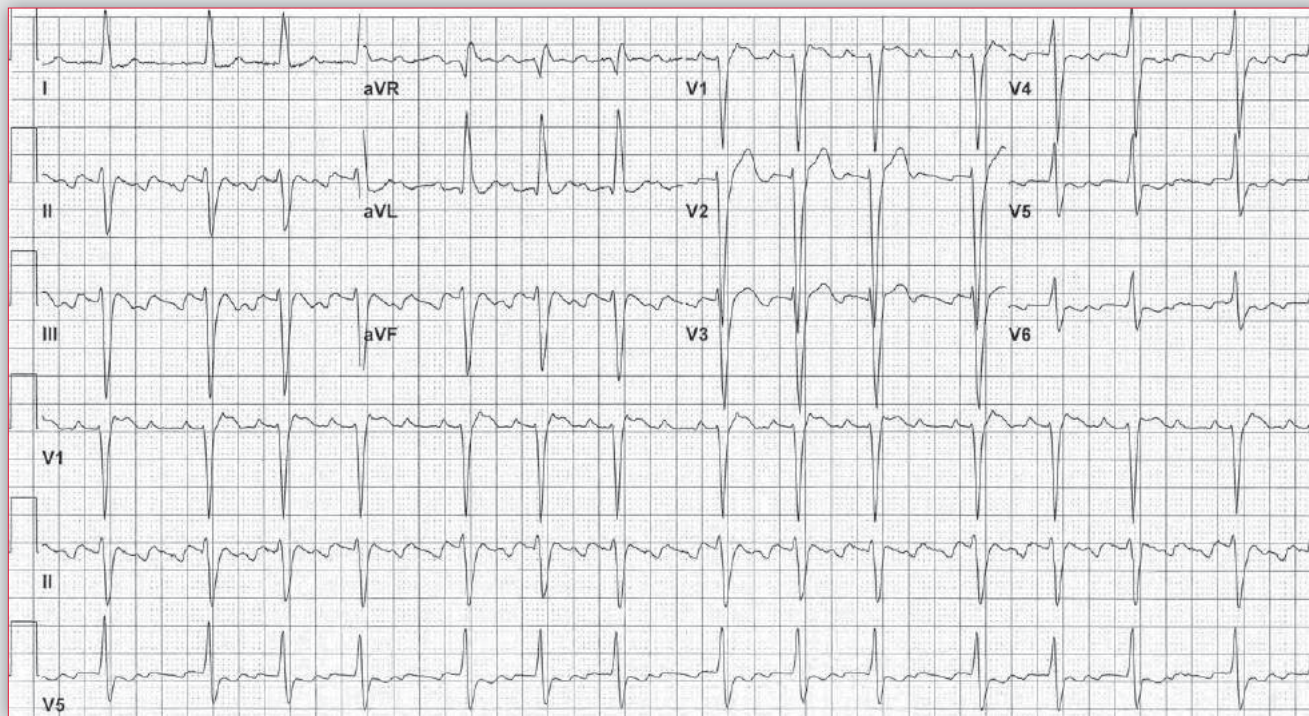
The patient's history of a preceding upper respiratory infection followed by throat pain (in this case, likely thyroid pain mistakenly interpreted as a "sore throat") and symptoms consistent with hyperthyroidism (sinus tachycardia, diaphoresis, and feelings of panic) suggest an acute (de Quervain's) thyroiditis. Serum thyroid hormone levels and TSH should be assayed. ■

Practice Case 98

A 73-year-old man with no known cardiac disease presents with acute onset of palpitations and dyspnea in the past 2 hours. An ECG is obtained (ECG 98A).

Since the symptoms began 2 hours prior to presentation (and hence < 48 hours), you attempt to chemically cardiovert the patient with propafenone using a dose of 450 mg (pill in

ECG 98A



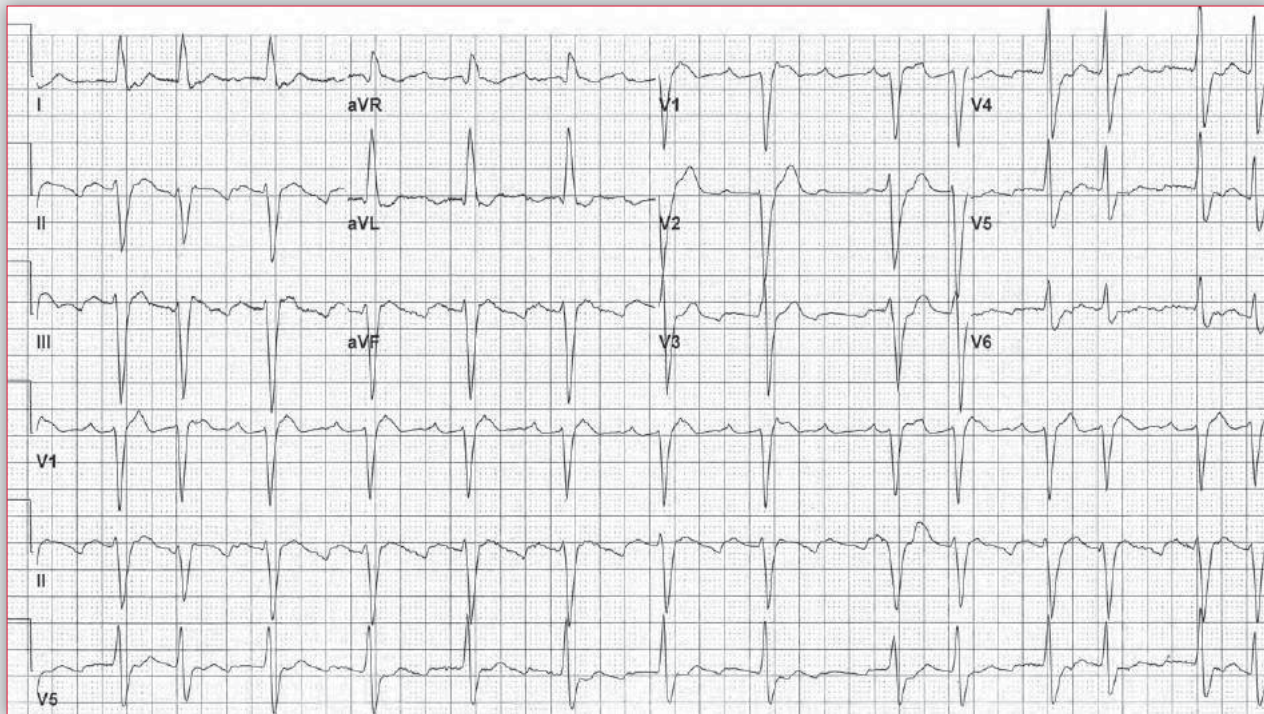
Practice Case 98

the pocket approach). Three hours after the administration of propafenone, another ECG is obtained (ECG 98B).

Shortly thereafter, the patient gets up from

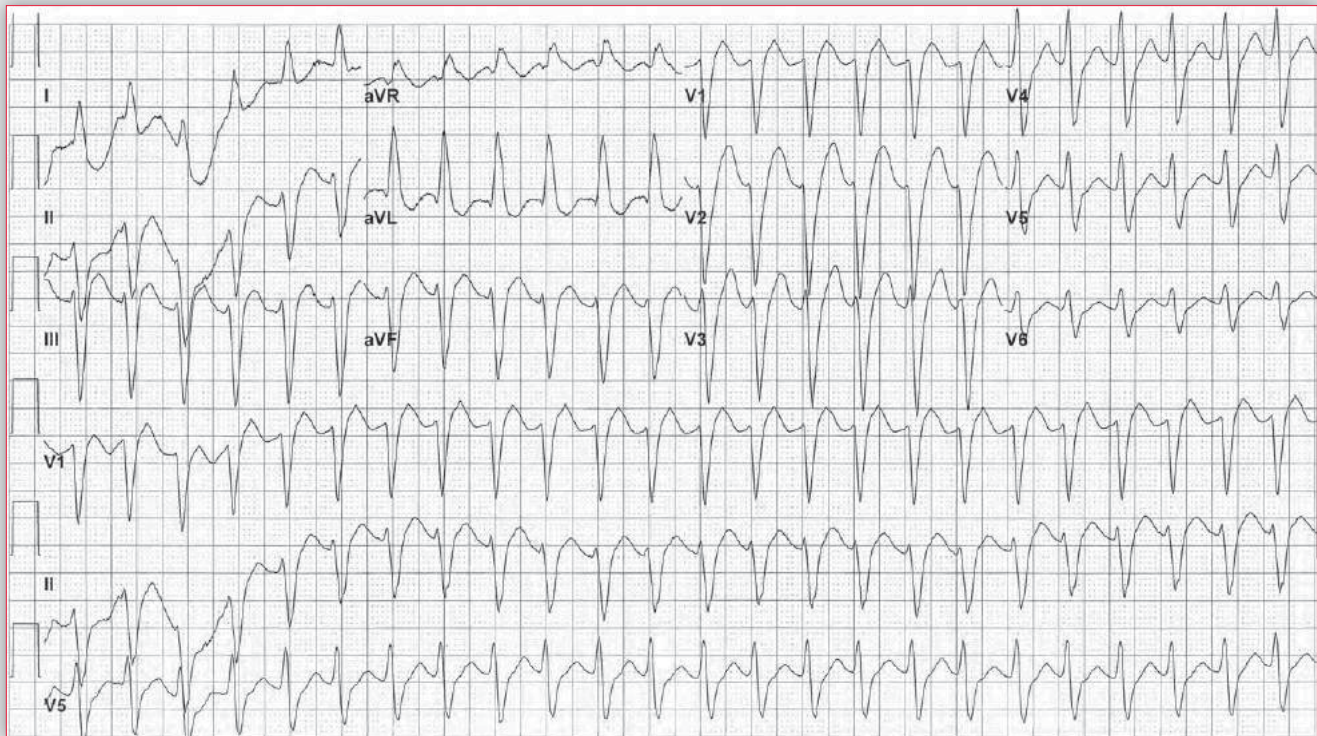
bed to go to the bathroom. The nurse notes that there is a marked change in the rhythm and QRS complexes on telemetry, and a rapid response is called. Another ECG is obtained (ECG 98C).

ECG 98B



Practice Case 98

ECG 98C



Practice Case 98

What is the rhythm abnormality in ECG 98A?

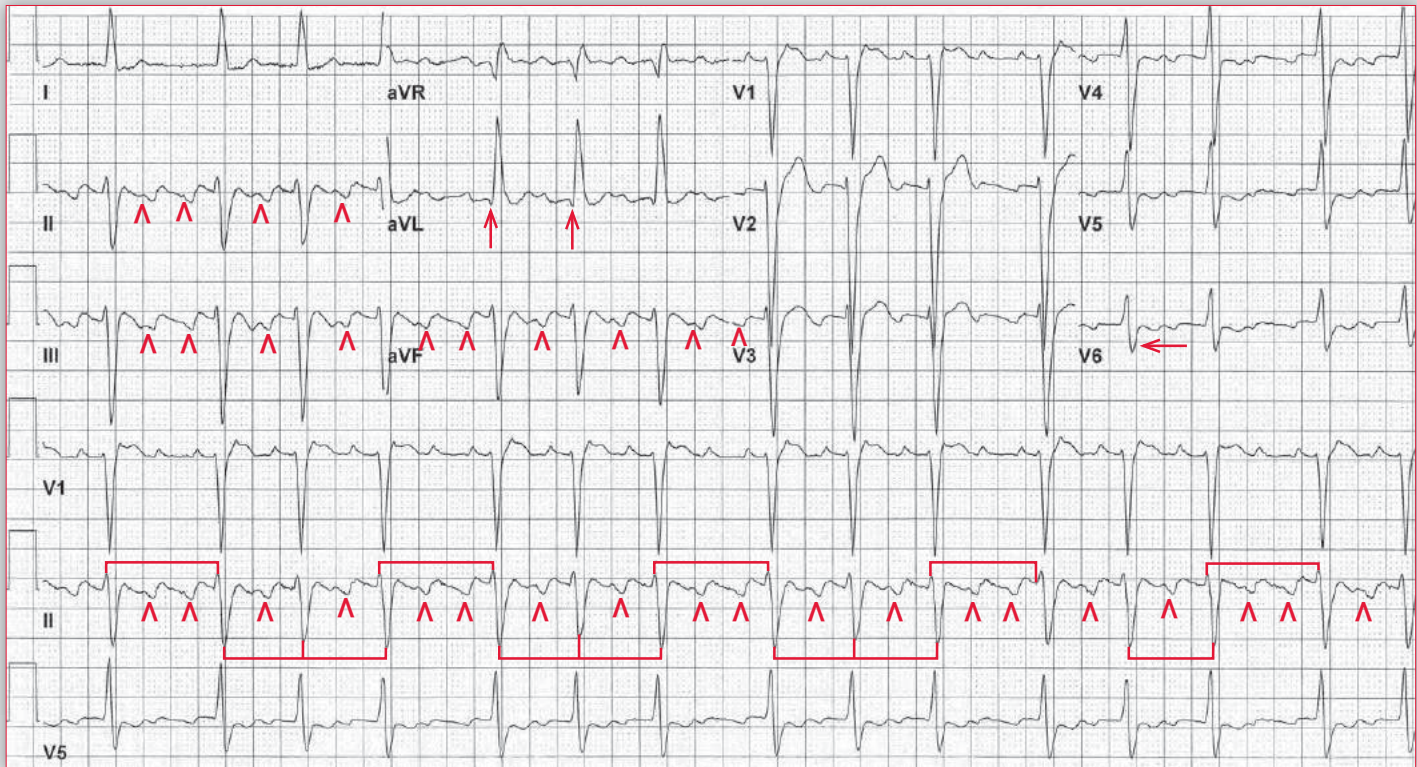
What is the rhythm in ECG 98B?

What explains the different ventricular rates in ECG 98B?

What is the underlying rhythm in 98C?

What is the reason for the change in rate and the change in QRS complex?

Podrid's Real-World ECGs

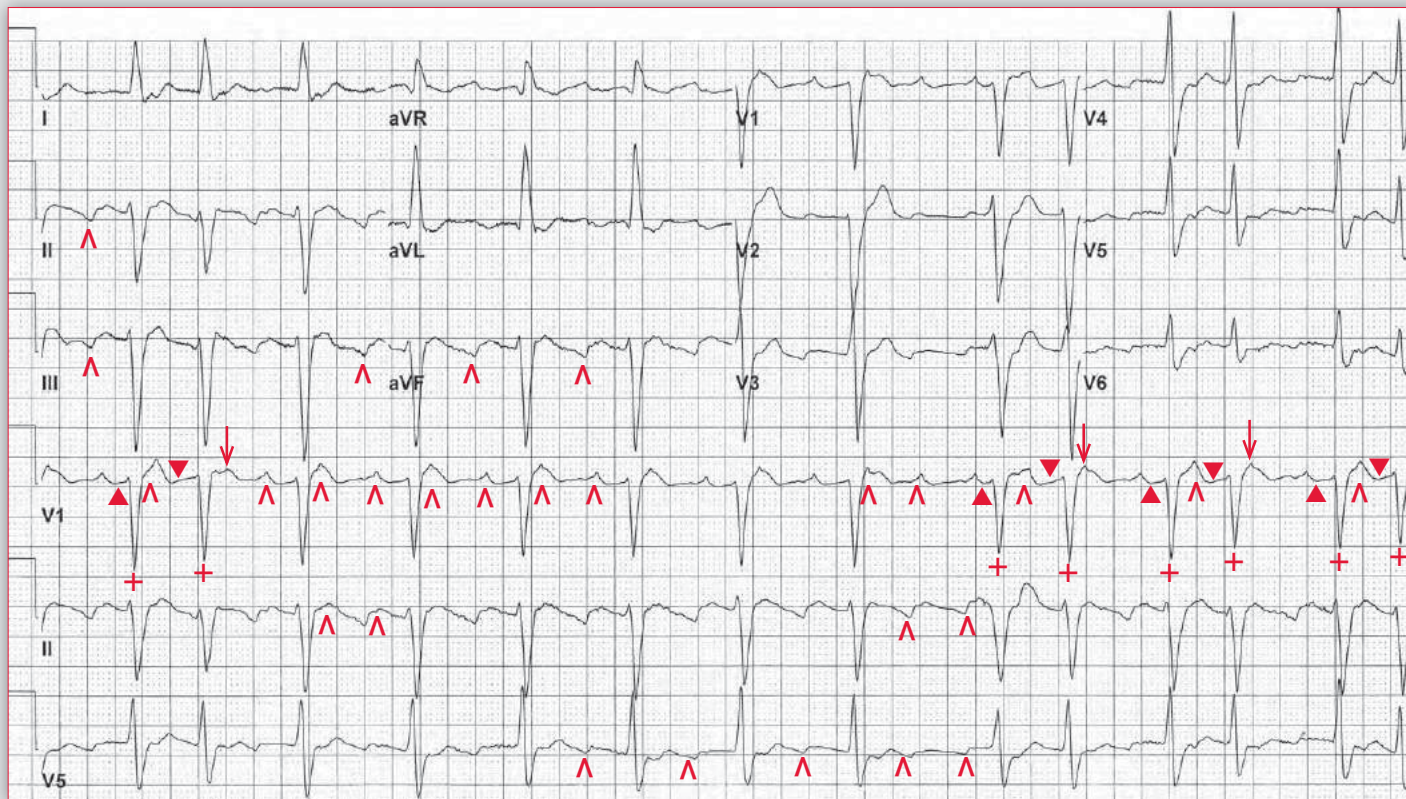


ECG 98A Analysis: Atrial flutter with variable AV conduction (2:1 and 3:1), intraventricular conduction delay, left anterior fascicular block

ECG 98A shows a regularly irregular rhythm at an average rate of 90 bpm. The irregularity is the result of short RR intervals (\sqcup), all of which are the same, and several longer RR intervals (\sqcap) that are all identical. There is evidence of atrial activity (\wedge) seen during the longer RR intervals, especially in leads II and V1. It can be seen that the atrial activity is regular at a rate of 240 bpm. In lead II, the atrial waveforms have continuous electrical activity, which is undulating (sawtooth pattern) without any isoelectric baseline between the waveforms. The waveforms are negative-positive in leads II, III, and aVF. This is atrial flutter with 2:1 and 3:1 AV conduction, although the flutter rate is slightly slower than is usually seen with atrial flutter (*ie*, atrial rate 260 to 320 bpm). The QRS complex duration is prolonged (0.12 sec), and the morphology resembles a left bundle branch block. However, there is a Q wave in lead aVL (\uparrow). There are no septal forces seen in a left bundle

branch block as the septal activation is via a septal (median) branch of the left bundle, which is not functional in a left bundle branch block. In addition, there is a terminal S wave in lead V6 (\leftarrow) that indicates the terminal forces are directed from left to right, more typical of a right bundle branch block. Left-to-right forces are not seen with a left bundle branch block as all the forces are directed from right to left. Hence this is an intraventricular conduction delay. The axis is extremely leftward, between -30° and -90° (positive QRS in lead I and negative in lead II and aVF with an rS morphology). As there is no left bundle branch block present, the extreme left axis is a left anterior fascicular block. The QT/QTc intervals are slightly prolonged (380/465 msec), but are normal when the prolonged QRS complex duration is considered (360/430 msec).

continues

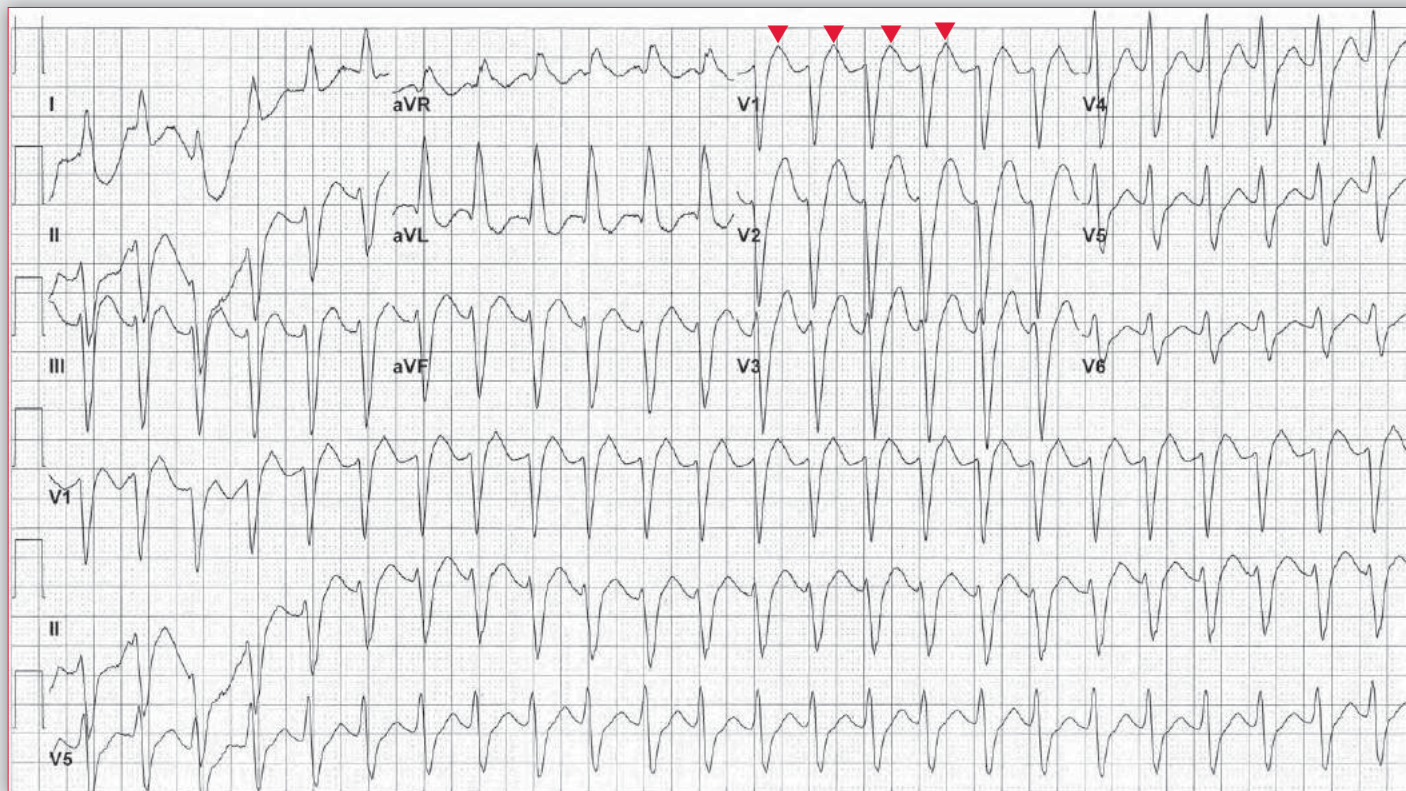


ECG 98B Analysis: Atrial flutter (slower atrial rate) with variable AV conduction and 3:2 Wenckebach, intraventricular conduction delay, left anterior fascicular block

ECG 98B is from the same patient as ECG 98A and was obtained three hours after receiving a loading dose of propafenone. The rhythm is still regularly irregular but the rate is slightly slower at 84 bpm. Regular atrial waves are still seen (^), but the atrial rate is slower at a rate of 150 bpm. However, the waveforms maintain a morphology typical for atrial flutter, *ie*, negative-positive waves in leads II, III, and aVF that are undulating (sawtooth) without an isoelectric baseline between the waveforms. AV conduction is variable primarily 2:1 and 3:1 AV conduction. However, there are several instances where there is a different pattern of the RR intervals (+) and hence AV conduction (*ie*, complexes 1–2, 9–10, 11–12, and 13–14). It can be seen that the flutter wave–QRS complex interval before complexes 1, 9, 11, and 13 (▲) are the same as all of the other flutter–QRS complex intervals. However, the flutter wave–QRS complex interval noted before complexes 2, 10, 12, and 14 (▼) is longer, while the flutter wave after complexes 2, 10, 12, and 14 is nonconducted (↓). This is a pattern of 3:2 Wenckebach.

The QRS complex duration, morphology, and axis are the same as in ECG 98A. The QT/QTc intervals are the same. Hence this is still atrial flutter with a slower atrial rate. The slowing of the atrial flutter rate is an effect of the class IC drug propafenone. The class I agents slow the rate of the rapid sodium influx into the heart during the onset of depolarization (*ie*, phase 0), thereby slowing the upstroke velocity of phase 0. This upstroke velocity determines the rate of impulse conduction; thus there is a slowing of velocity of depolarization in both atrial and ventricular myocardium. Therefore, the rate of impulse conduction within the circuit responsible for atrial flutter is decreased, slowing the flutter rate. As a result of the slower atrial rate, there is the potential for less AV block, as the AV node is being stimulated at a slower rate, allowing for more impulses to get through. This likely accounts for the occurrence of 3:2 Wenckebach rather than fixed 2:1 and 3:1 conduction.

continues



ECG 98C Analysis: Atrial flutter (slower rate) with 1:1 AV conduction, intraventricular conduction delay with use-dependent effect, left anterior fascicular block

Several minutes after ECG 98B was obtained, the patient began to walk around his room while still on telemetry. A rapid response was called based on observation of a change in heart rate and QRS complexes noted by the nurse on telemetry. ECG 98C was obtained as soon as the patient returned to his bed. There is a regular rhythm at a rate of 150 bpm. There are no obvious P waves seen, *ie*, no obvious atrial activity, although there is a sharp-pointed T wave in lead V1, suggesting a superimposed P wave (▼). However, the ventricular rate is identical to the atrial flutter rate seen in ECG 98B. Hence this is atrial flutter with 1:1 conduction. The QRS complex is now diffusely wider (0.16 sec) compared to that seen in ECGs 98A and 98B, although it has the same morphology of an intraventricular conduction delay.

As seen in ECG 98B, the atrial flutter rate slowed from 240 bpm to 150 bpm as a result of the class IC antiarrhythmic drug. Although the atrial rate remained at 150 bpm in ECG 98C, there are several explanations for the occurrence of 1:1 AV conduction. Since the normal, unstimulated AV node is capable of conducting impulses at rates of up to about 170 to 180 bpm, the slower atrial flutter rate increases the potential for 1:1 AV conduction and hence acceleration of the ventricular response rate. In addition, at the slower rate of atrial flutter, there is a reduction in the amount of concealed conduction within the AV node, with an increased AV conduction velocity. Lastly, in this case, there was also an increase in AV nodal conduction velocity as a result of sympathetic stimulation, as occurs with activity.

The class I agents, especially the class IC drugs including propafenone, exhibit a property known as use dependency. In this situation, the effect of the drug on slowing of the upstroke of phase 0 of the action potential is more pronounced at faster heart rates. This is due to the fact that normally the drug binds to a receptor in the sodium channel, resulting in a slowing in the rate of sodium influx during phase 0 of the action potential. This is the sodium-channel blocking activity, and since the velocity of phase 0 determines the impulse conduction velocity, the sodium-channel blocker will produce a slowing of impulse conduction, observed as a widening of the QRS complex. These agents generally dissociate in diastole. When the heart rate is increased, there is less time for dissociation and hence the number of receptors that are blocked increases, causing a reduction in the rate of sodium influx during phase 0 and hence a further rate-related slowing of impulse conduction velocity, which produces a rate-related widening of the QRS complex duration. It is important to recognize this effect, as the rate-related widening of the QRS complex can be confused with the occurrence of ventricular tachycardia, particularly when P waves or evidence of atrial activity are not seen, as in this case. ■

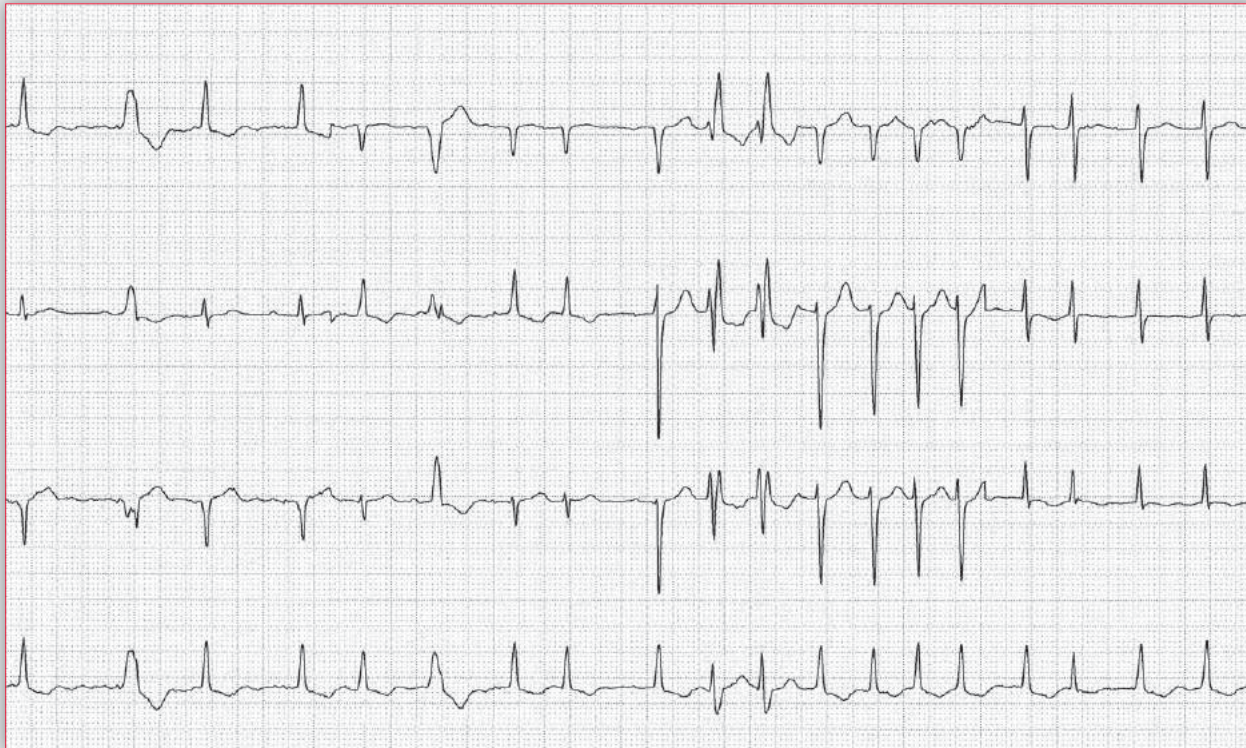
Notes

Practice Case 99

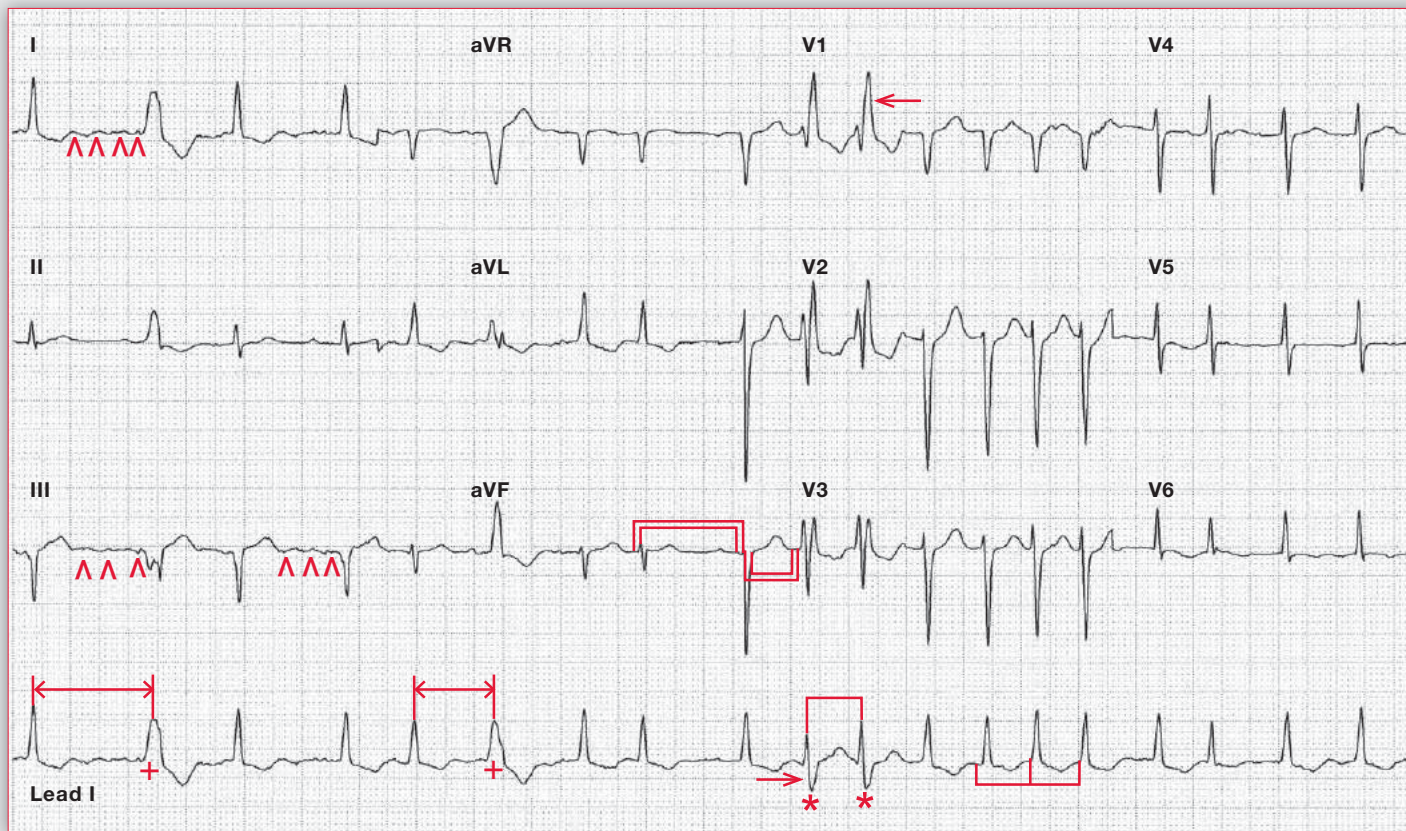
A 74-year-old woman with known heart failure with preserved ejection fraction (*ie*, diastolic heart failure) presents with worsening dyspnea in the setting of palpitations. The following ECG is obtained.

What is the rhythm abnormality?

What is/are the mechanism(s) for the wider QRS complexes in the ECG?



Podrid's Real-World ECGs



ECG 99 Analysis: Atrial fibrillation, Ashman phenomenon, premature ventricular complexes (possibly ventricular parasystole)

ECG 99 shows an irregularly irregular rhythm at a rate of approximately 126 bpm. There are no obvious P waves seen before or after any QRS complex. There are low-amplitude undulations of the baseline (\wedge), especially obvious in leads I, II, and III. The three rhythms that are irregularly irregular include sinus arrhythmia (with one P-wave morphology and one PR interval); multifocal atrial rhythm (rate < 100 bpm), or multifocal atrial tachycardia (rate > 100 bpm), which have > 3 different P-wave morphologies with no dominant P-wave morphology and variable PR intervals; and atrial fibrillation (in which there are no definable P waves). Hence the rhythm is atrial fibrillation. Most of the QRS complexes have a normal duration (0.08 sec) and have a leftward axis between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (300/430 msec).

There are several QRS complexes that are wide (0.14 sec) (+,*), *ie*, the second, sixth, tenth, and eleventh complexes. The second and sixth QRS complexes (+) have a left bundle branch block morphology (broad R wave in leads I and aVF). They have a variable coupling interval (\leftrightarrow) between the narrow QRS complex and the wide QRS complexes. Moreover, the coupling interval is longer than that seen with the shorter RR intervals. Hence these QRS complexes do not manifest rate-related aberration; rather, they are premature ventricular

complexes. Since both premature ventricular complexes have the same QRS morphology, they are unifocal. The variability in the coupling intervals of these unifocal premature ventricular complexes suggests ventricular parasystole. In this situation, the mechanism responsible for the premature ventricular complexes is enhanced activity of an ectopic focus. The more common mechanism for premature ventricular beats is reentry. In this situation, the coupling interval between the premature ventricular complex and the preceding complex is constant.

The tenth and eleventh QRS complexes (*) have a right bundle branch block morphology with an RSR' (\leftarrow) in lead V1 and a broad S wave in lead I (\rightarrow). Although the RR intervals associated with these complexes are short (\sqcap), the aberrancy is not rate-related as there are narrow (nonaberrated QRS complexes) that are associated with even shorter RR intervals (\sqcup). It can be seen that these two wide or aberrated complexes follow a combination of long-short RR intervals (\sqcap, \sqcup). This is an Ashman phenomenon.

The usual form of aberration, which is associated with faster heart rate (*ie*, rate-related), is due to conduction block resulting from underlying conduction system disease (and the inability of the conduction system to conduct at a faster rate). On the other hand, the aberration

continues

Notes

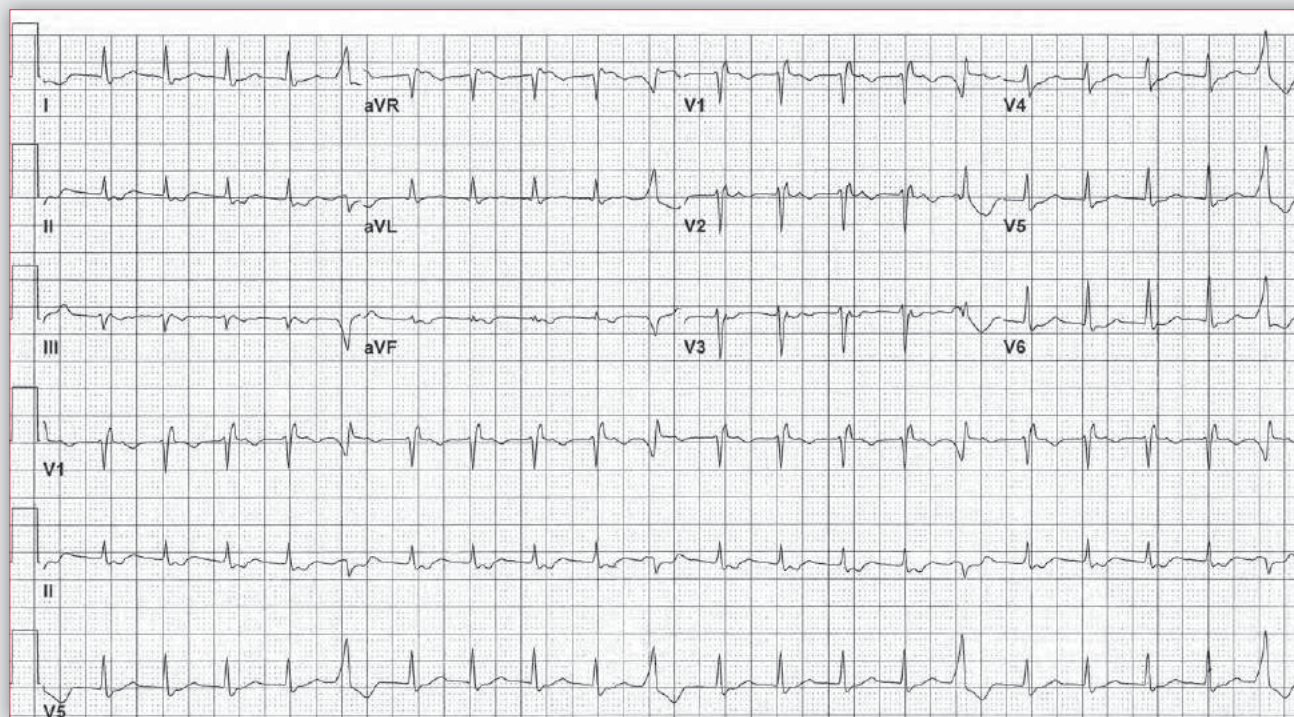
associated with the Ashman phenomenon is the result of normal physiologic changes in refractoriness of the His-Purkinje system (phase 3 block). At slower heart rates (longer RR interval), His-Purkinje refractoriness increases, while with faster heart rates (shorter RR interval) refractoriness decreases. Whenever there is an abrupt change in rate, *ie*, slower (long RR with longer refractoriness) to faster (short RR interval), refractoriness does not have time to adapt; hence there is blockage of the impulse conduction in a part of the His-Purkinje system, resulting in an aberration. The Ashman phenomenon is most often associated

with a right bundle branch block, likely because the refractoriness of the right bundle is longer than that of the left bundle. The Ashman phenomenon is most often seen in atrial fibrillation, which is associated with marked irregularity in RR intervals and more episodes of long-short RR intervals. However, the Ashman phenomenon can be seen whenever there is an abrupt change in heart rate, going from slow to fast. The Ashman phenomenon may also last for several QRS complexes. As seen with this ECG, it lasts for two consecutive beats. ■

Practice Case 100

A 70-year-old man with a recent permanent pacemaker placement for sick sinus syndrome (set to a ventricular rate of 40) presents with palpitations that started while climbing a flight of stairs during in-patient

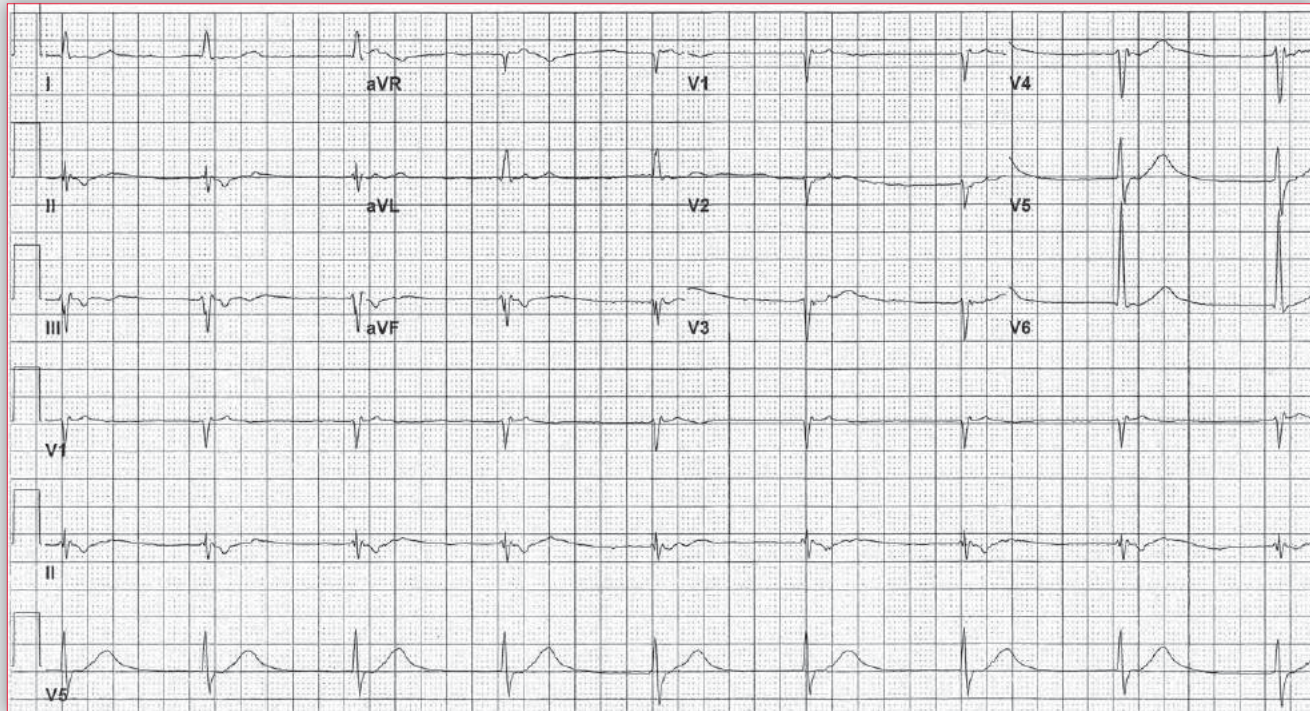
ECG 100A



Practice Case 100

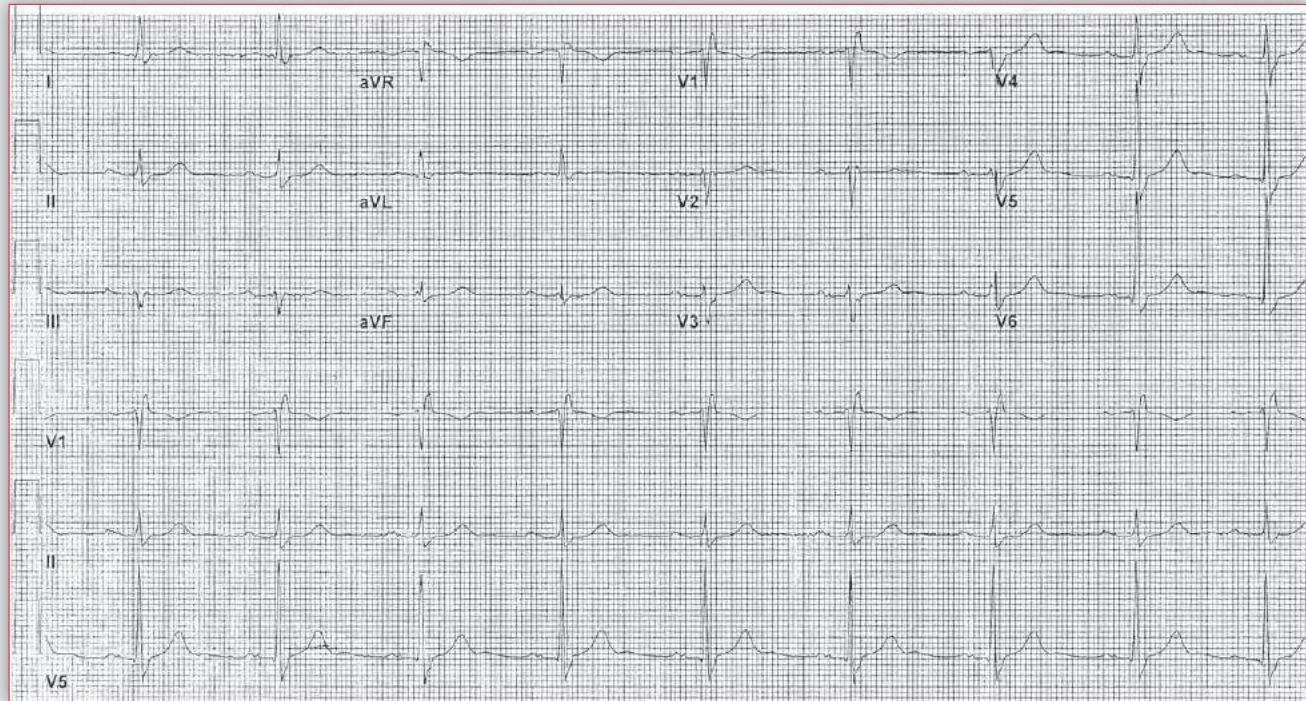
rehabilitation. An ECG was obtained (ECG 100A). After treatment with metoprolol, the patient's symptoms improve and a second ECG is obtained (ECG 100B). These ECG are compared to the patient's baseline ECG (ECG 100C).

ECG 100B



Practice Case 100

ECG 100C

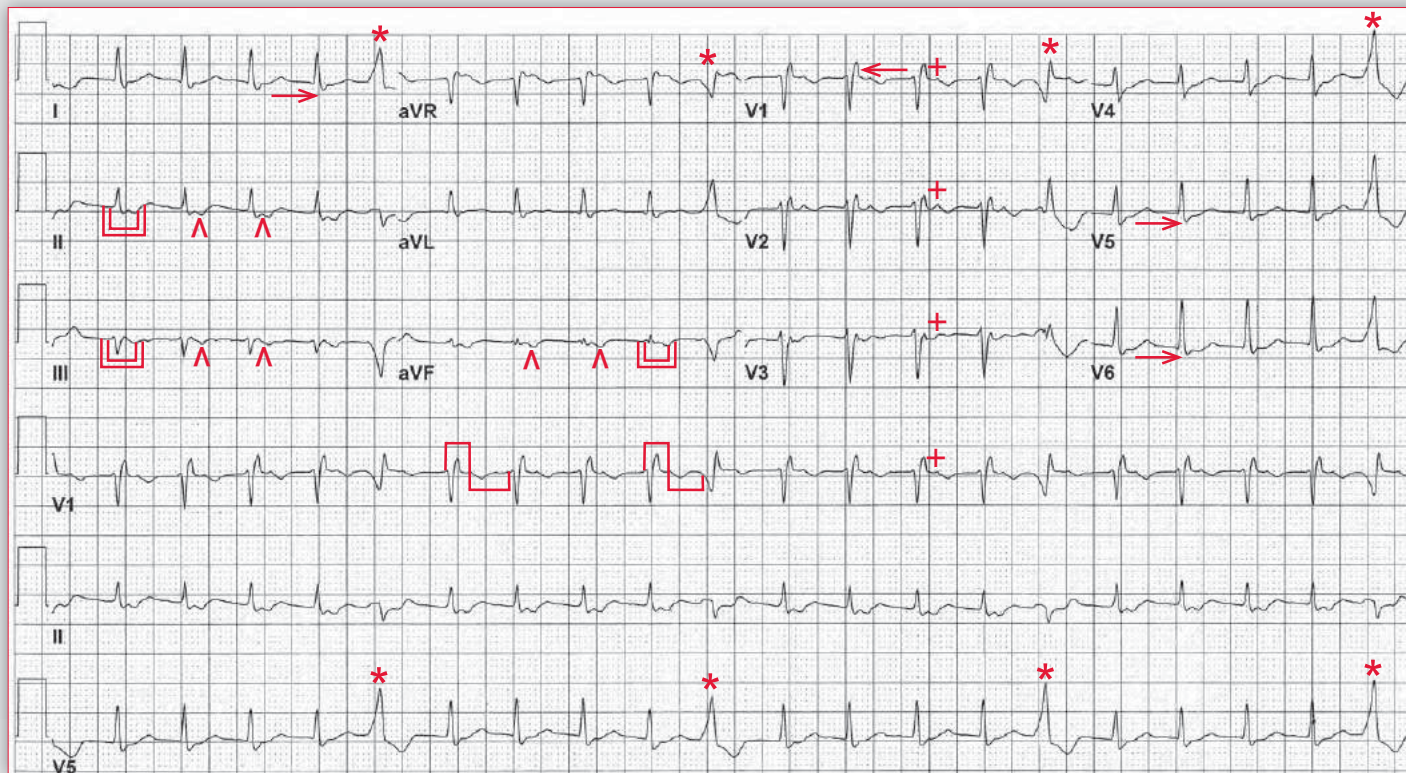


Practice Case 100

What is the rhythm abnormality in ECG 100A?

Do ECGs 100B and 100C help establish a diagnosis? If so, what is the arrhythmia?

Podrid's Real-World ECGs



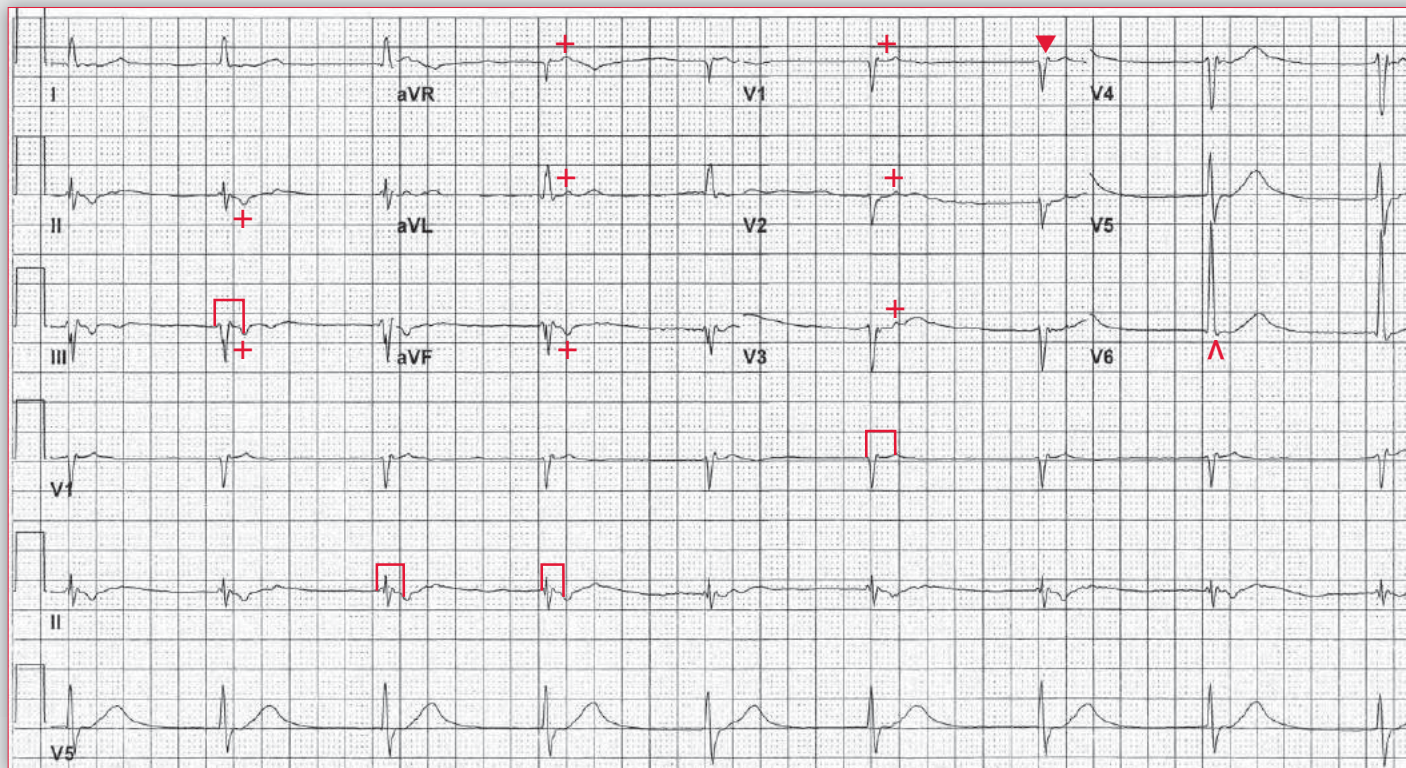
ECG 100A Analysis: Supraventricular tachycardia, short RP tachycardia, ectopic junctional tachycardia, intraventricular conduction delay to right ventricle, premature ventricular complexes, low voltage

ECG 100A mostly shows a regular rhythm at a rate of 130 bpm. There are four complexes (fifth, tenth, fifteenth, and twentieth) (*) that are premature (slightly shorter RR interval). These QRS complexes are wide (0.16 sec) and have a different QRS morphology (*) compared to the majority of the QRS complexes. These are premature ventricular complexes. Most of the QRS complexes have a duration slightly less than 0.12 second. They have a normal axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QRS complex morphology resembles a right bundle branch block with an RSR' in lead V1 (\leftarrow) and terminal S waves in leads I and V5–V6 (\rightarrow). However, the QRS complex width is less than usually seen with a right bundle branch block; hence this is an intraventricular conduction delay to the right ventricle (often termed incomplete right bundle branch block). In addition there is low voltage (*ie*, < 5 mm in each limb lead and < 10 mm in each precordial lead). The QT/QTc intervals are normal (300/440 msec and 280/400 msec when corrected for the prolonged QRS complex

duration). There are no P waves seen before any QRS complex. However, there are P waves (+) seen after each QRS complex, especially in V1–V3. There is a short RP interval (0.20 sec) (\square) and long PR (0.32 sec) (\sqcup). Using these intervals, it can be seen that the negative waves (^) after the QRS complexes in leads II, III, and aVF have the same RP interval (\sqcup); they are negative P waves. Hence, this is a short RP tachycardia. Etiologies include sinus tachycardia (which can be eliminated as the P waves are negative in leads II and aVF), atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, atrioventricular reentry tachycardia, or an uncommon form of a common or typical atrioventricular nodal reentrant tachycardia, known as slow-slow. In this situation, the slow pathway conducts the impulse antegradely to the ventricles, while the fast pathway that conducts the impulse retrogradely to the atrium conducts relatively slowly as a result of age-related changes or the effect of a drug.

continues

Podrid's Real-World ECGs



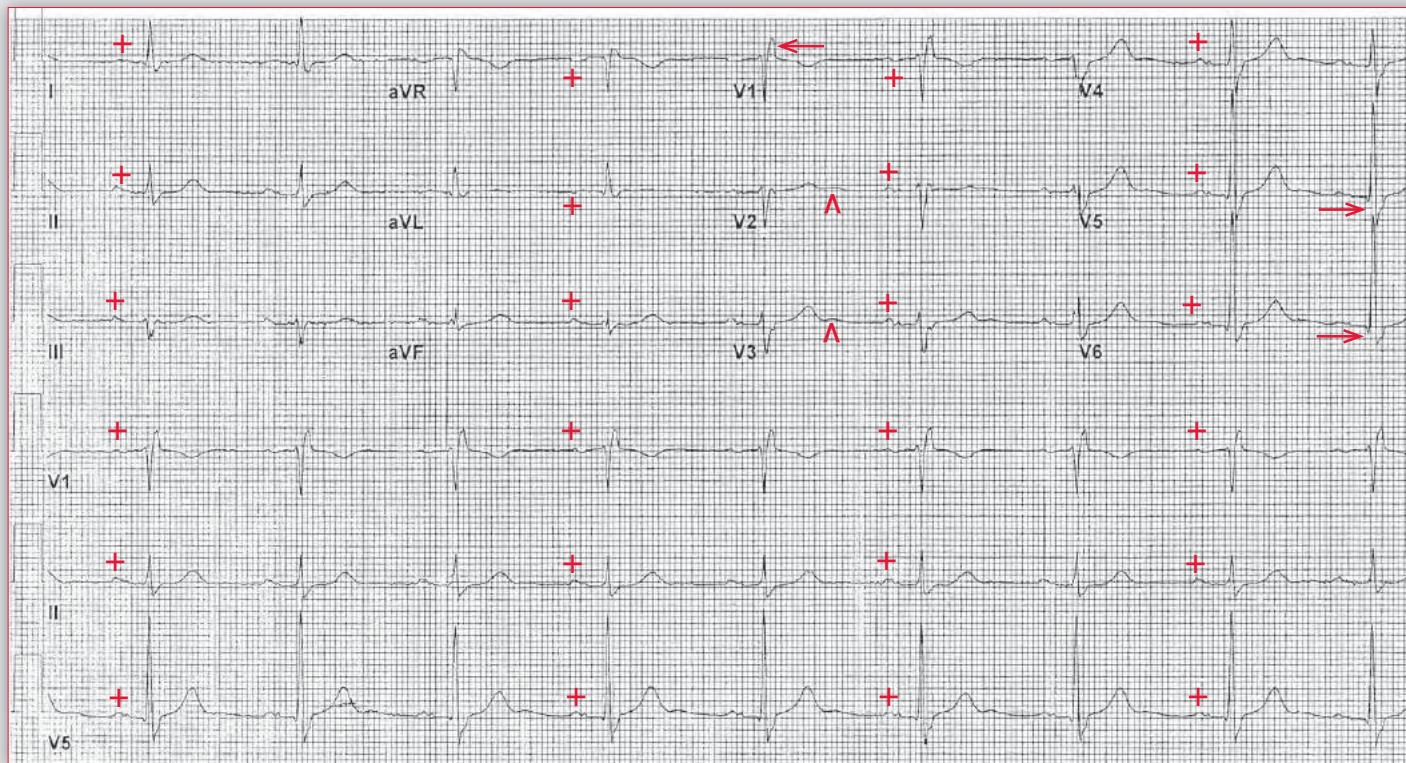
ECG 100B Analysis: Ectopic junctional rhythm, intraventricular conduction delay to right ventricle

ECG 100B is from the same patient as ECG 100A. There is a regular rhythm at a rate of 50 bpm. The QRS complex duration is normal (0.08 sec) and the intraventricular conduction delay is less obvious, although there is still an R' in lead V1 (▼) and a small S wave in lead V6 (^). The fact that the QRS complex is narrower and the right bundle branch like pattern is not seen, is due to the fact that there was a rate-related intraventricular conduction delay seen in ECG 100A. The axis is now leftward between 0° and -30° (positive QRS complex in leads I and II and negative in aVF). The QT/QTc intervals are normal (480/440 msec). There are no P waves seen before any QRS complex,

but there are P waves (+) seen after the QRS complex, most apparent in leads V1–V3 as well as in leads II, III, aVR, aVL, and aVF (^), where it is negative. The RP interval (□) is constant, and it is the same as the RP interval seen in ECG 100A (*ie*, 0.20 sec). Hence this is an ectopic junctional rhythm with retrograde P waves due to retrograde atrial activation. Therefore, the rhythm in ECG 100A is an ectopic junctional tachycardia. An ectopic junctional tachycardia is identical to an ectopic junctional rhythm, except that the rate is > 100 bpm. Importantly, retrograde VA conduction with an ectopic junctional rhythm is the same regardless of rate.

continues

Podrid's Real-World ECGs



ECG 100C Analysis: Normal sinus rhythm, first-degree AV block, intraventricular conduction delay to right ventricle, U waves in V2–V3

ECG 100C is from the same patient as ECGs 100A and 100B. There is a regular rhythm at a rate of 54 bpm. The QRS complex duration is prolonged (almost 0.12 sec), and there is a RSR' morphology in lead V1 (←) and a terminal broad S wave in leads I and V5–V6 (→). The morphology resembles a right bundle branch block, but the QRS complex is not wide enough to be a complete right bundle branch block. Hence this is an intraventricular conduction delay to the right ventricle. The QRS complex morphology is the same as the QRS complex in ECG 100A. As the rate is slightly faster than in ECG 100B (QRS complex that is narrower without an IVCD), this further supports the fact that the conduction delay is rate-related. The axis is the same as in ECG 100B. The QT/QTc intervals are normal (430/420 msec).

There is a P wave (+) before each QRS complex with a stable PR interval (0.30 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm with a first-degree AV block. When comparing this ECG with ECG 102A and ECG 102B, there is no P wave seen after the QRS complex. There is a U wave (^) seen in leads V2–V3. ■

Notes

Practice Case 101

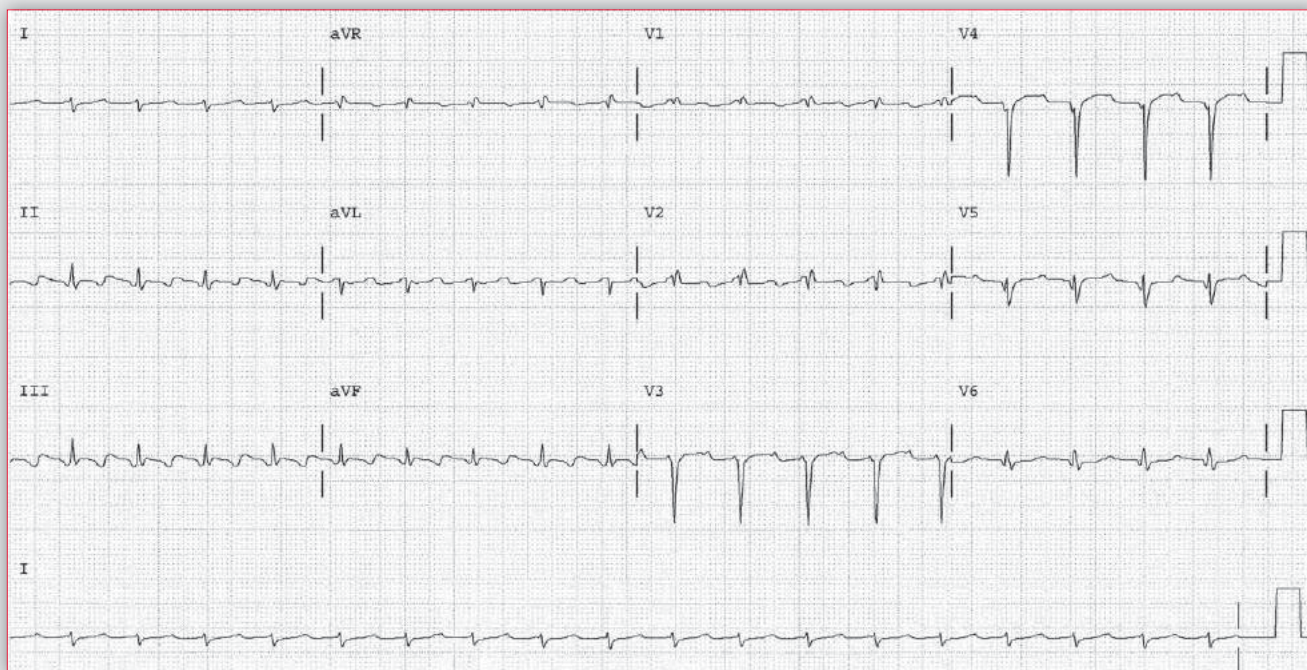
A 79-year-old woman with known coronary artery disease and recent symptoms of an upper respiratory infection presents to the emergency room with dizziness, near syncope, and palpitations. On physical examination, her heart rate is 120 bpm and regular, blood pressure is 85/55, and oxygen saturation is 98% on room air. You note a jugular venous pulsation at the angle of the jaw

(> 12 cm) and significant jugular venous distention. There are no crackles in the lung fields. Cardiac sounds are distant. The following ECG is obtained.

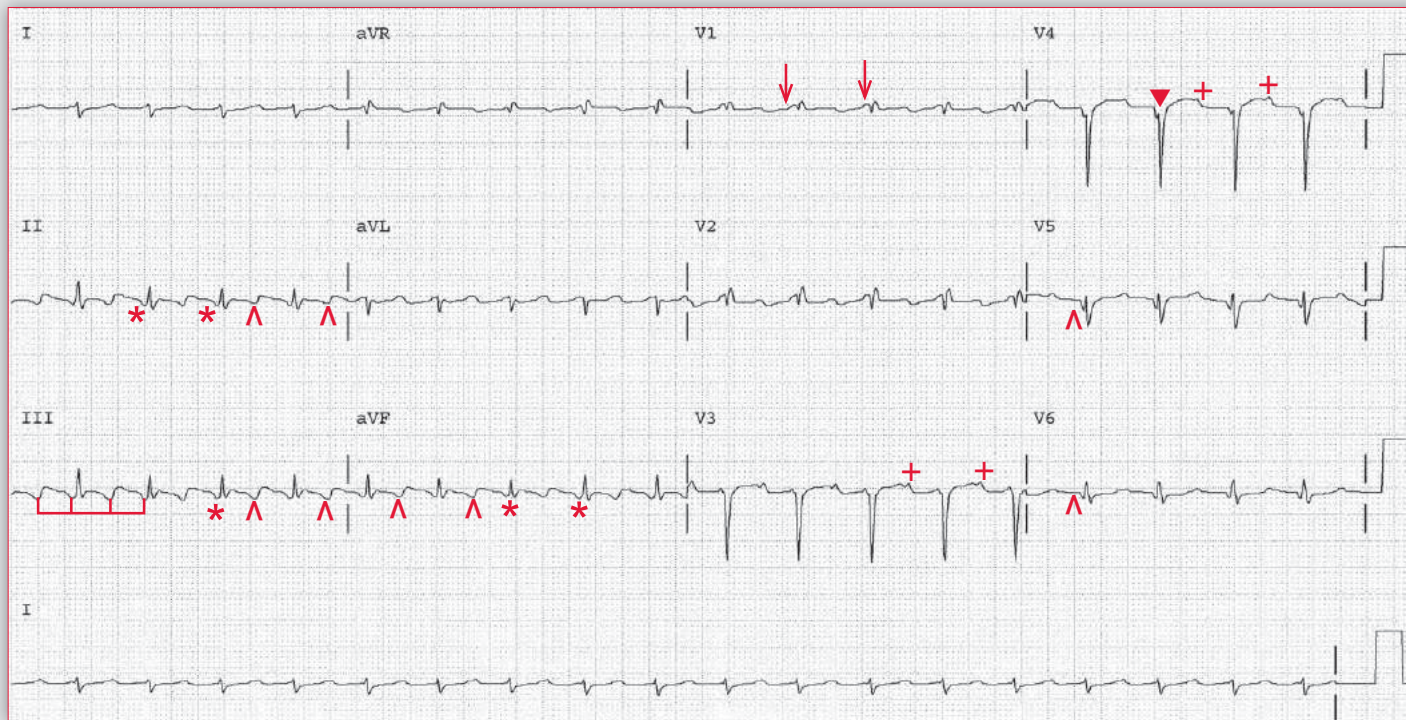
In order to follow her hemodynamics more closely, you place a radial arterial catheter for blood pressure monitoring. Peak arterial waveform measurements vary with respiration and range from 78 mm Hg to 92 mm Hg.

What is the rhythm abnormality?

What is the clinical diagnosis and your next step in management?



Podrid's Real-World ECGs



ECG 101 Analysis: Atrial flutter with 2:1 AV conduction, left posterior fascicular block, low voltage in the limb leads, old anteroapical and anterolateral wall myocardial infarction

ECG 101 shows a regular rhythm at a rate of 120 bpm. The QRS complex duration is normal (0.08 sec) and there is low voltage in the limb leads (< 5 mm in each lead). The axis is rightward between $+90$ and $+180$ degrees (negative QRS complex in lead I and positive in lead aVF). This is a left posterior fascicular block. Noted are QS complexes in leads V3–V4 (▼) and significant Q waves in V5–V6 (▲), diagnostic of an old anteroapical and anterolateral wall myocardial infarction.

There are no obvious P waves seen. However, there are positive deflections (+) seen on the T waves in leads V3–V4, which are atrial waveforms. Based on the timing, the negative waveforms (▲) in leads II, III, and aVF are not T waves, but are the atrial waveforms. This would be called a short RP tachycardia. However, a similar negative waveform (*) can be seen just before the QRS complex in leads II, III, and aVF. These are not Q waves, but represent a second atrial waveform. These atrial waveforms are regular (□) at a rate of 260 bpm. Although the QRS complex appears to have an RSR' morphology in leads V1–V3, the first deflection (↓) is actually an atrial waveform. As there is a regular atrial rate of 260 bpm, the rhythm is atrial flutter with 2:1 AV conduction. In addition, the morphology of the atrial waveforms in

leads II, III, and aVF appear to have continuous undulations without any isoelectric baseline. The QT/QTc intervals are difficult to establish because of the atrial flutter waves.

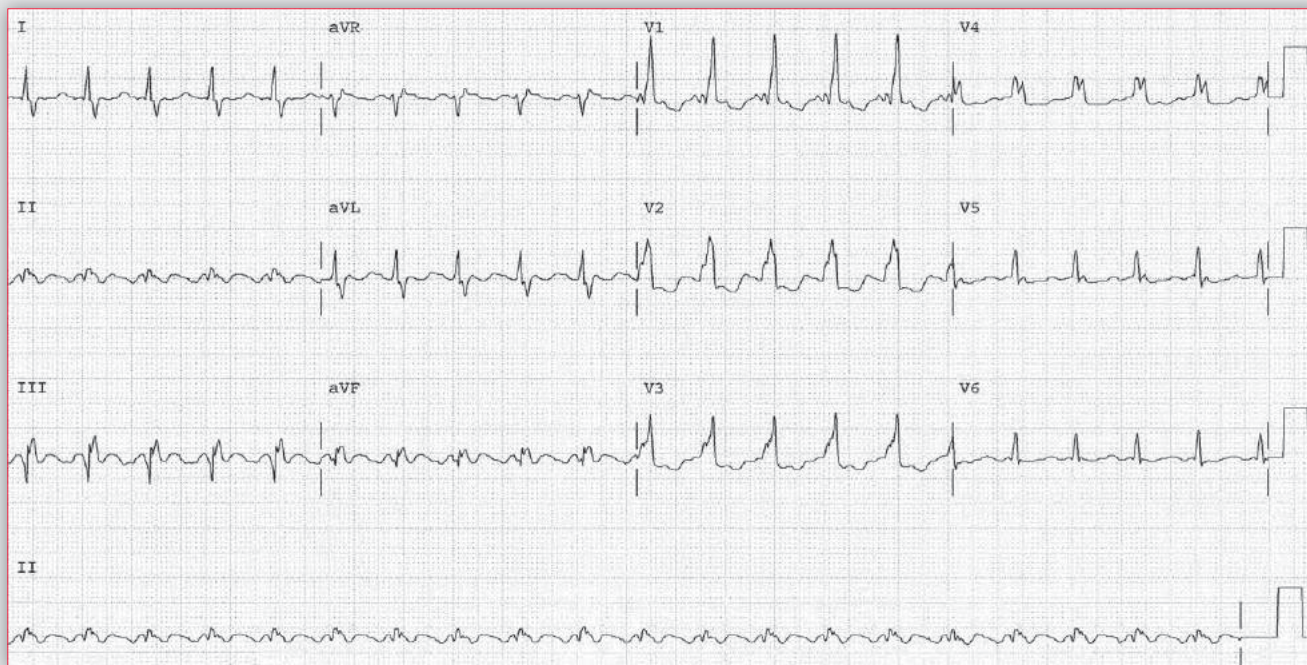
Elevated jugular venous pressure, hypotension, and distant heart sounds constitute Beck's triad and suggest the presence of cardiac tamponade. This is further supported by the presence of low voltage in the ECG limb leads. QRS or electrical alternans, which may be seen with tamponade, is not present. There is also pulsus paradoxus, which is defined as a decrease in systolic blood pressure by > 10 mm Hg with inspiration. The placement of a radial arterial catheter facilitates assessment for pulsus paradoxus. The patient should be given intravenous fluids and undergo drainage of the pericardial effusion. An echocardiogram should be obtained first (if the patient is stable enough to wait) to identify the size and location of the effusion, in order to guide percutaneous drainage. It is possible that her clinical presentation does not indicate the severity of the pericardial effusion as a result of the atrial flutter. The rapid atrial rate and reduced atrial contraction could contribute to worsened hemodynamics. ■

Practice Case 102

A 56-year-old man with known coronary artery disease and prior myocardial infarction presents with 4 hours of palpitations and dyspnea. He is noted to have a tachycardia, blood pressure of 90/60 and a PO_2 sat of 88%. His chest x-ray indicates vascular congestion.

An ECG is obtained (ECG 102A). The patient is urgently cardioverted because of the hypotension and evidence of heart failure. An antiarrhythmic drug is begun. ECG 102B is obtained 3 days later.

ECG 102A



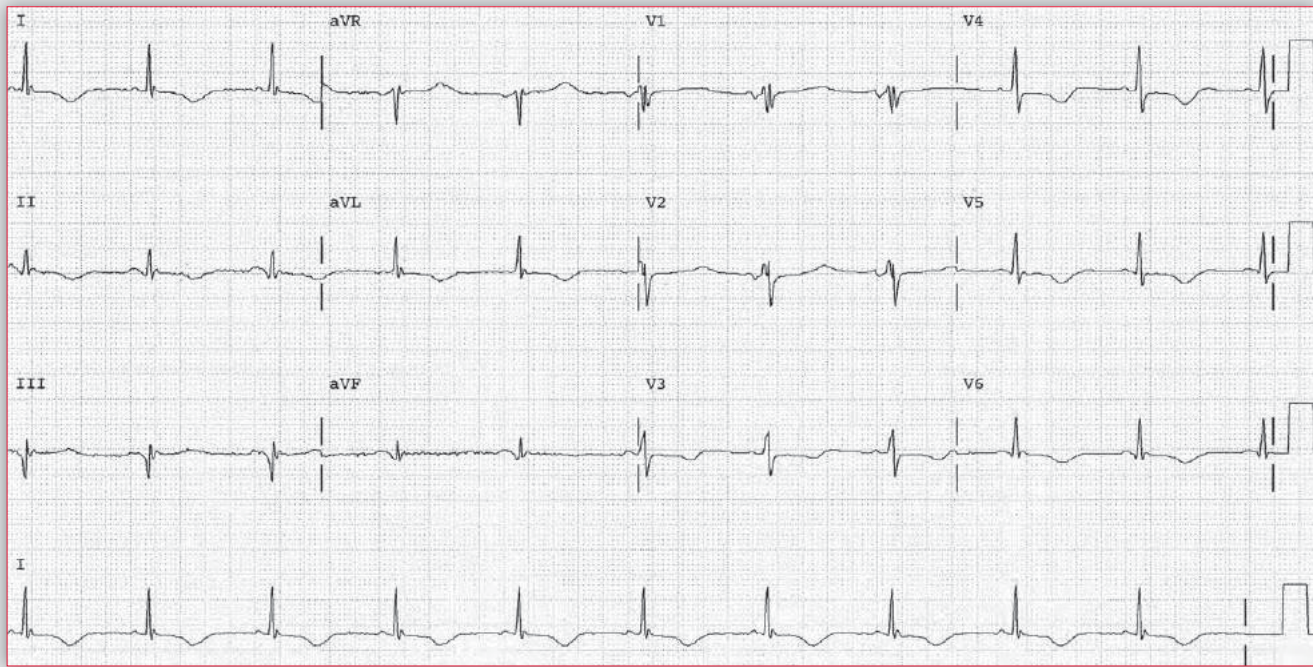
Practice Case 102

What is the etiology of the arrhythmia?

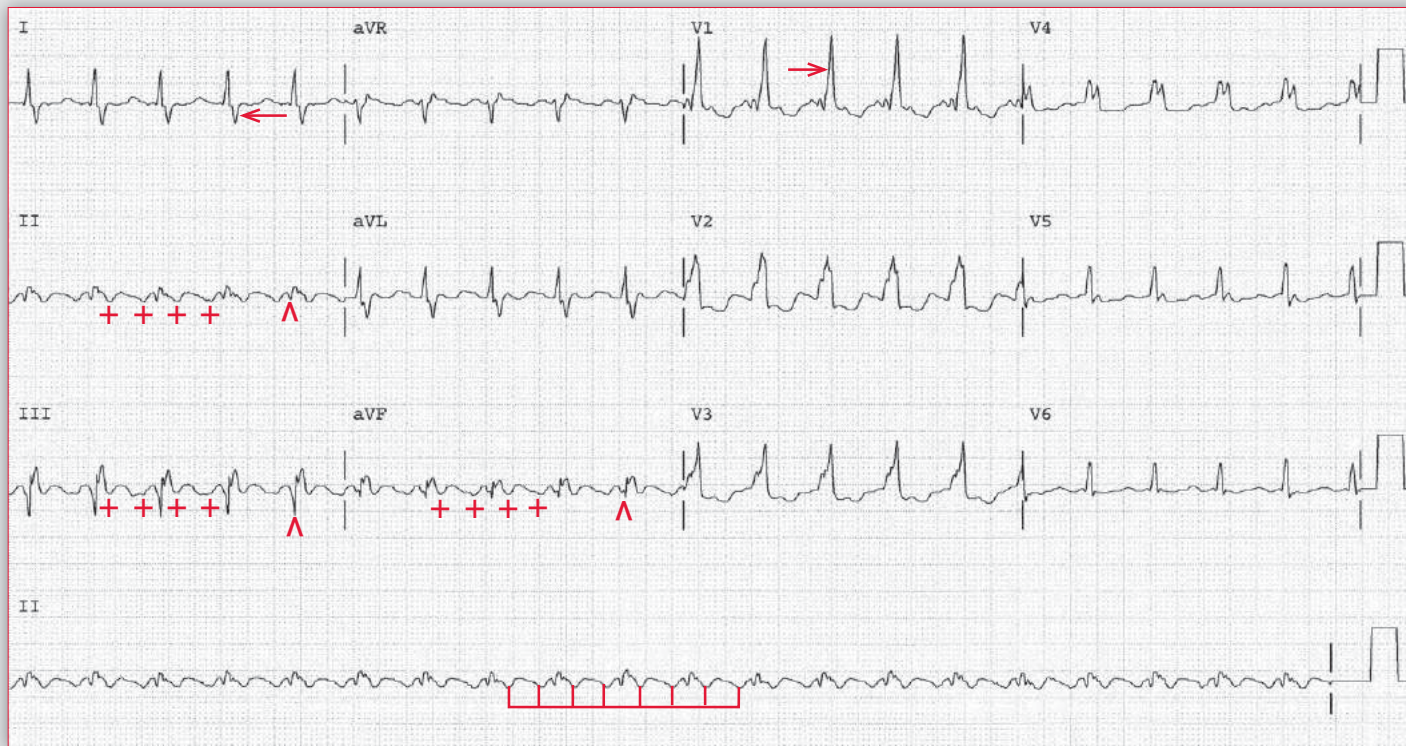
What accounts for the changes in the QRS complex?

What is the antiarrhythmic drug being administered?

ECG 102B



Podrid's Real-World ECGs



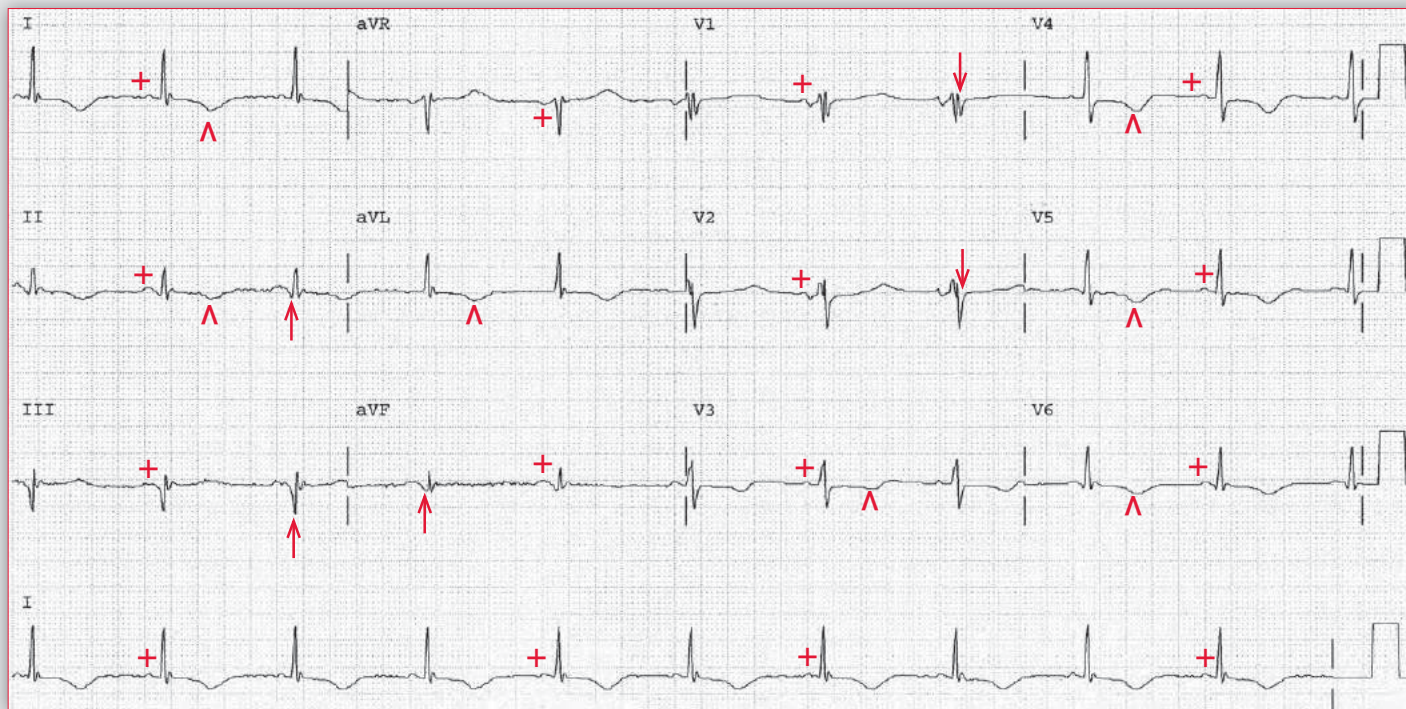
ECG 102A Analysis: Atrial flutter with 2:1 AV block, right bundle branch block, old inferior myocardial infarction

ECG 102A shows a regular rhythm at a rate of 130 bpm. The QRS complex duration is increased (0.14 sec), and there is a right bundle branch block morphology with an RSR' in lead V1 (→) and broad S wave in lead I (←). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). Hence this is a wide complex tachycardia. There is evidence of regular atrial activity (⏏), most obvious in

leads II, III, aVF, and lead V1 (+). The atrial rate is 260 bpm and there is continuous or undulating atrial activity. Hence the rhythm is atrial flutter with 2:1 AV conduction. Q waves are seen in leads II, III, and aVF (Λ) consistent with an old inferior wall myocardial infarction. The QT/QTc intervals are prolonged (340/510 msec) but are normal when the prolonged QRS complex duration is considered (300/440 msec).

continues

Podrid's Real-World ECGs



ECG 102B Analysis: Normal sinus rhythm, old inferior myocardial infarction, nonspecific T-wave abnormalities, QT prolongation

ECG 102B is from the same patient as ECG 102A and it was obtained after 3 days of antiarrhythmic drug therapy. There is a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec), and there is a normal axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). Slight notching of the QRS complex (↓) is seen in leads V1 and V2, suggesting an intraventricular conduction delay. There are Q waves in leads II, III, and aVF (↑), characteristic of an old inferior wall myocardial infarction. However, a right bundle branch block morphology is no longer present. Hence ECG 102A showed atrial flutter with 2:1 AV block and a rate-related right bundle branch block; this accounted for the wide complex tachycardia.

There are T-wave abnormalities or inversions (^) seen in leads I, II, aVL, and V3–V6. The positive T wave in lead aVR is actually T-wave inversion. The QT/QTc intervals are prolonged (520/520 msec).

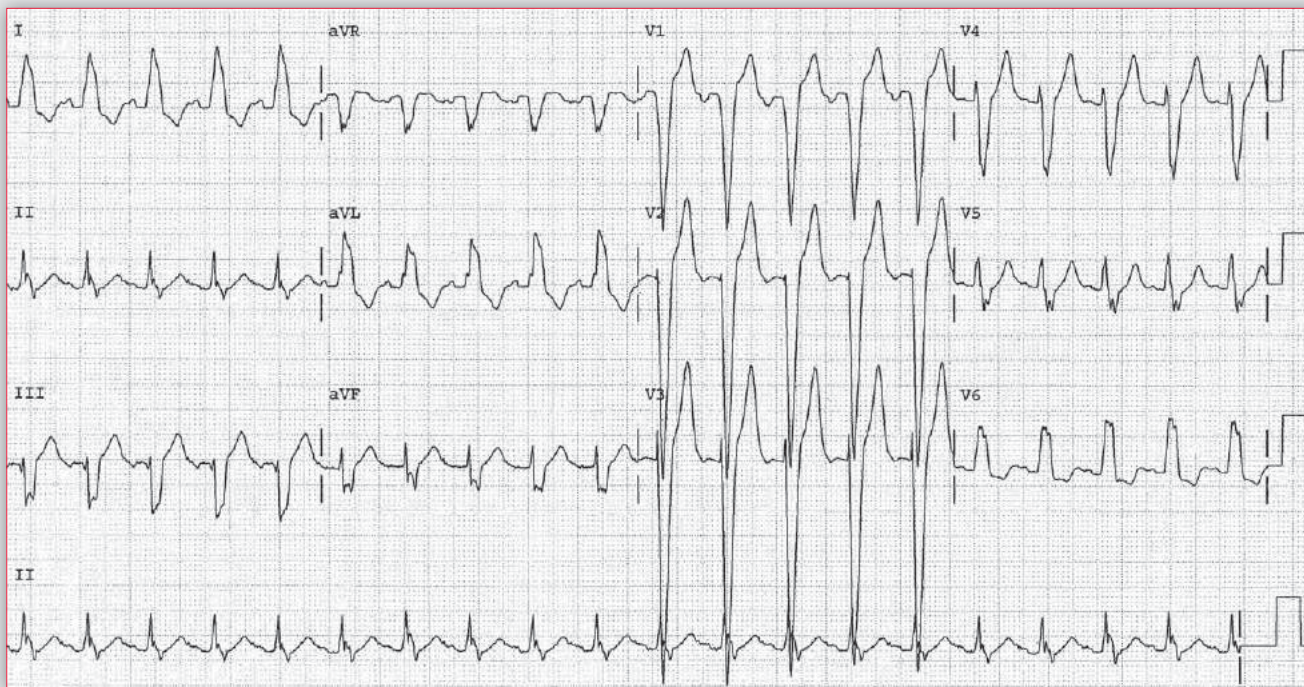
The presence of prolonged QT/QTc intervals as a result of an antiarrhythmic drug is consistent with a class III antiarrhythmic drug effect. This class of antiarrhythmic drugs works by preventing the efflux of potassium during phase 3 of the action potential. As a result, there is prolongation of the refractory period that is manifest on the surface ECG as the QT interval. There are several class III antiarrhythmic agents being used as therapy for atrial flutter, including ibutilide (which is only available for intravenous use), amiodarone (which also has class I, II, and IV antiarrhythmic effects), sotalol (which also has class II or β -blocking effects) and dofetilide (which has only class III antiarrhythmic effects). The QT/QTc interval can also be increased with a class IA drug (intravenous procainamide, quinidine, or disopyramide). However, these agents are rarely prescribed. Although it is not completely certain, it is most likely that the patient was begun on therapy with sotalol, which generally requires 2 to 3 days of therapy before steady-state blood levels are achieved and QT prolongation is seen. Amiodarone usually requires several weeks of loading before therapeutic blood levels are achieved and QT prolongation is observed. Current guidelines recommend a reduction in dose if the corrected QT interval exceeds 500 msec. This patient's QT interval will need to be monitored and the patient will need to avoid other QT prolonging agents. ■

Practice Case 103

A 42-year-old man with no known cardiac risk factors presents with acute onset substernal chest pressure to the emergency department. His blood pressure is elevated at 165/95 mm Hg, but his examination is otherwise unremarkable. An ECG is obtained (ECG 103A).

Within minutes after your initial treatment, the patient states that the chest pain resolved and a repeat ECG is obtained (ECG 103B).

ECG 103A



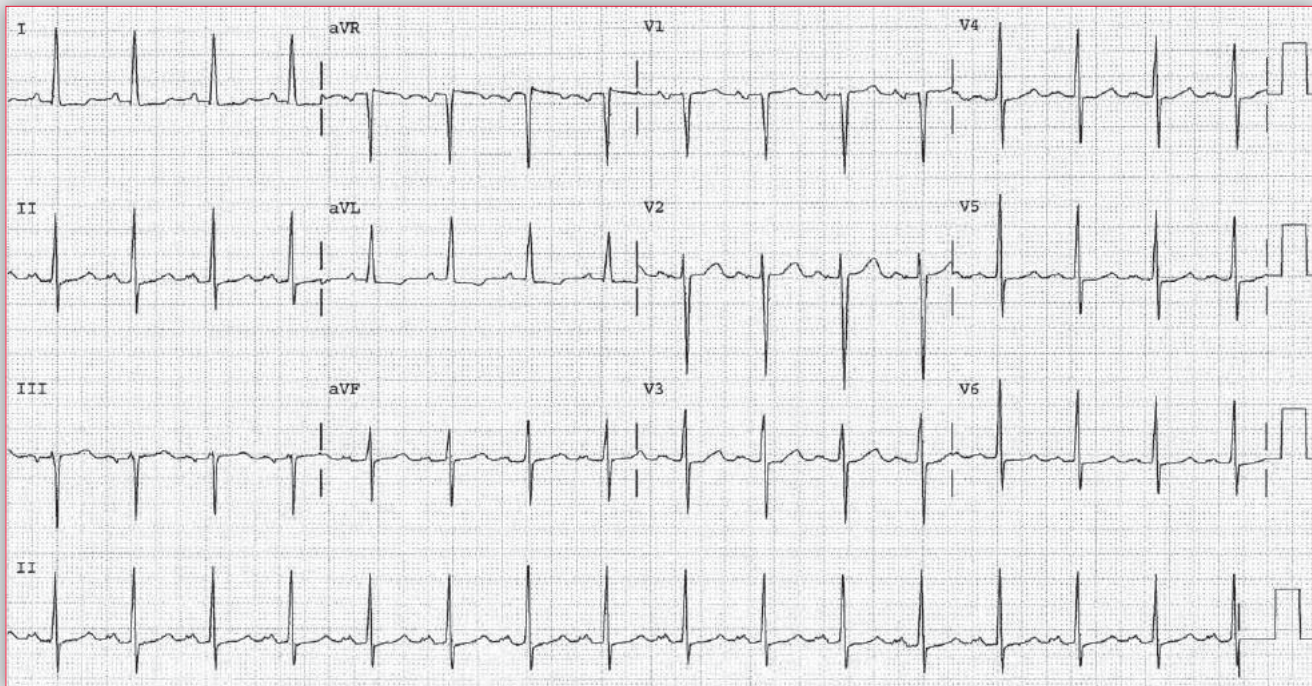
Practice Case 103

What are the ECG 103A abnormalities?

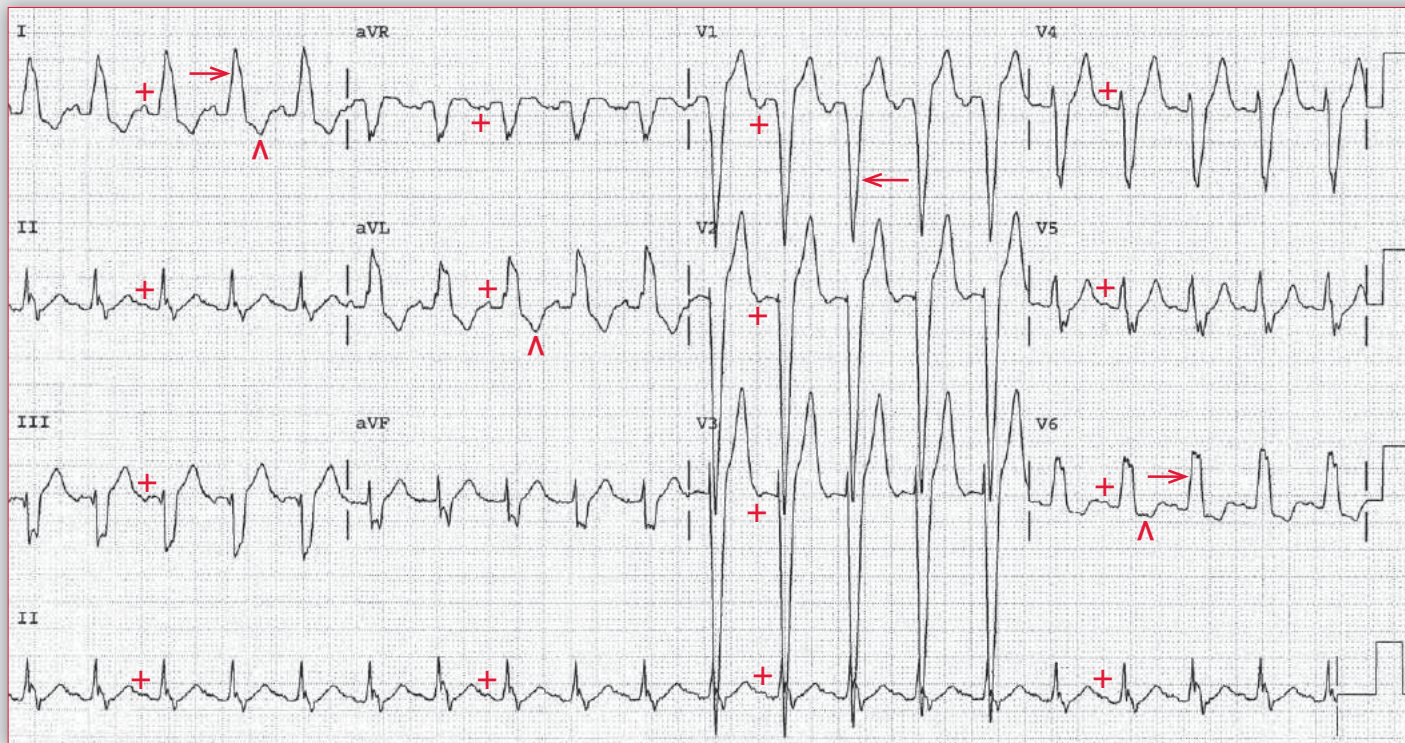
What is your next step in management?

How does ECG 103B change your management?

ECG 103B



Podrid's Real-World ECGs



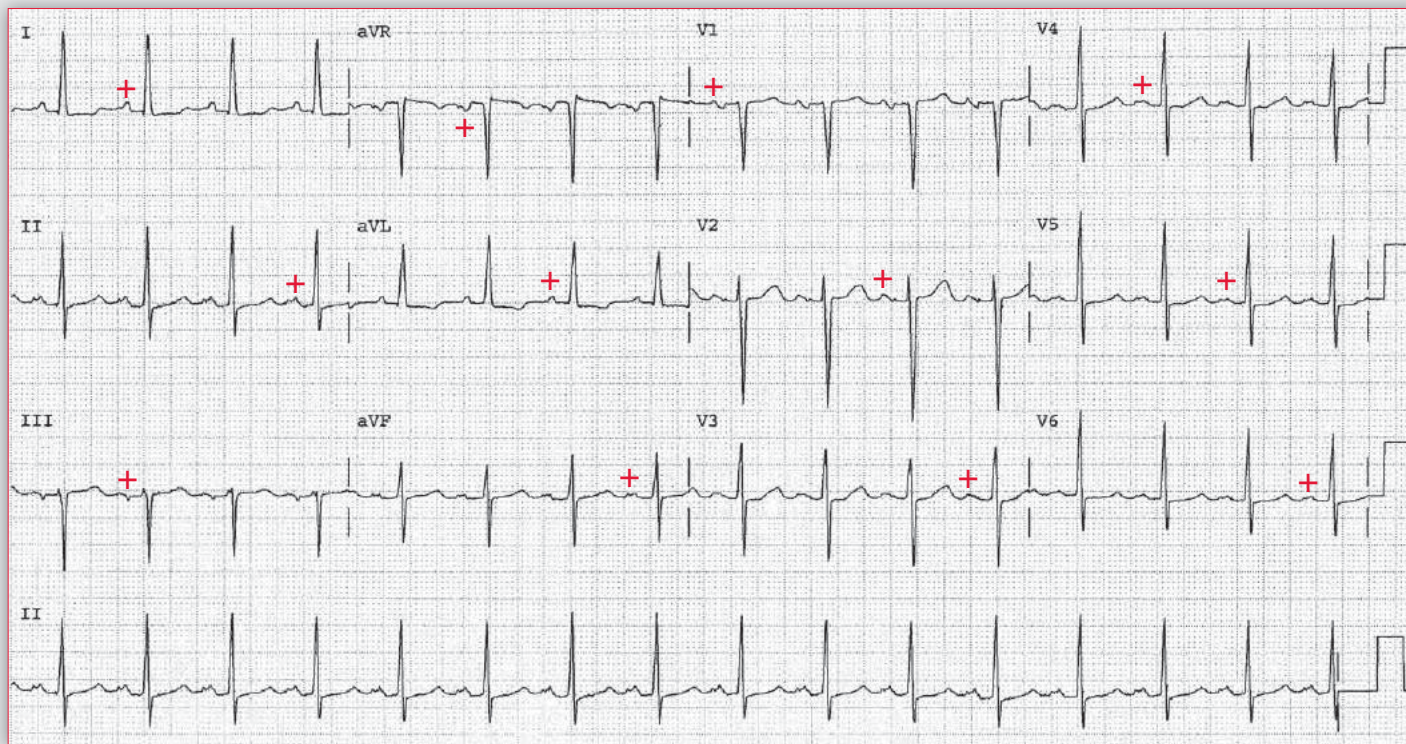
ECG 103A Analysis: Sinus tachycardia, left atrial hypertrophy, rate-related left bundle branch block with associated ST-T wave changes

ECG 103A shows a regular rhythm at a rate of 120 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, and V4–V6. Hence this is a sinus tachycardia. The P wave is primarily negative in leads V1–V2, consistent with left atrial hypertrophy. The QRS complex duration is increased (0.16 sec), and there is a left bundle branch block (LBBB) morphology with a broad R wave in leads I and V6 (→) and deep QS complex in lead V1 (←). There are ST-T wave changes that are associated with the left bundle branch block (^). The axis is leftward, between 0° and –30° (positive QRS complex in lead I and II and negative in leads aVF). The QT/QTc intervals are prolonged (360/530 msec), but are normal when the prolonged QRS complex duration is considered (300/450 msec).

Active ischemia or acute myocardial infarction cannot be reliably identified in a patient with a left bundle branch block as left ventricular activation bypasses the normal His-Purkinje system and there is direct myocardial activation. In this situation, therapy depends upon the clinical history and presentation of the patient. Up to 30% of patients with an LBBB pattern presenting with chest pain are ultimately determined to have an acute MI. This patient with LBBB and chest pain should be acutely treated with aspirin, anticoagulation, nitrates, and β -blocker therapy. Cardiac troponins should be evaluated and serial ECGs should be obtained, as dynamic changes in ST segments and/or T waves or new Q waves are suggestive of acute myocardial infarction in the presence of an LBBB. Based on the clinical presentation, consideration should be made for cardiac catheterization for possible reperfusion therapy.

continues

Podrid's Real-World ECGs



ECG 103B Analysis: Normal sinus rhythm with first-degree AV block, left atrial hypertrophy

ECG 103B is from the same patient as ECG 103A. The patient was treated with the medications as indicated above, including a β -blocker. There was relief of the chest pain and a subsequent ECG was obtained (ECG 103B). There is a regular rhythm at a rate of 94 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and this is a normal sinus rhythm with a first-degree AV block. The P wave is broad and notched in II, aVF and V2–V6, suggesting left atrial hypertrophy.

The QRS complex duration is normal (0.08 sec), and there is an axis of 0° (QRS positive in lead I and biphasic in lead aVF). The QRS morphology is normal. The QT/QTc intervals are normal (340/425 msec). Hence at a slower heart rate, the left bundle branch block is no longer present, confirming that the left bundle branch block seen in ECG 103A is a rate-related left bundle branch block.

As the QRS complex is now normal in duration without a left bundle, abnormalities affecting the left ventricle can be interpreted as left ventricular activation via the normal His-Purkinje system. There is no evidence for acute myocardial infarction on this patient's ECG and, therefore, there is no indication for urgently sending the patient for cardiac catheterization. Myocardial ischemia may still have been present at the fast heart rate when the patient first presented. With medical therapy, ischemia may have resolved, and with the repeat ECG changes of ischemia, *ie*, ST-segment depressions, may have also resolved. Therefore, based on the clinical suspicion for ischemia, further evaluation might be indicated, *ie*, a noninvasive evaluation with stress testing. ■

Notes

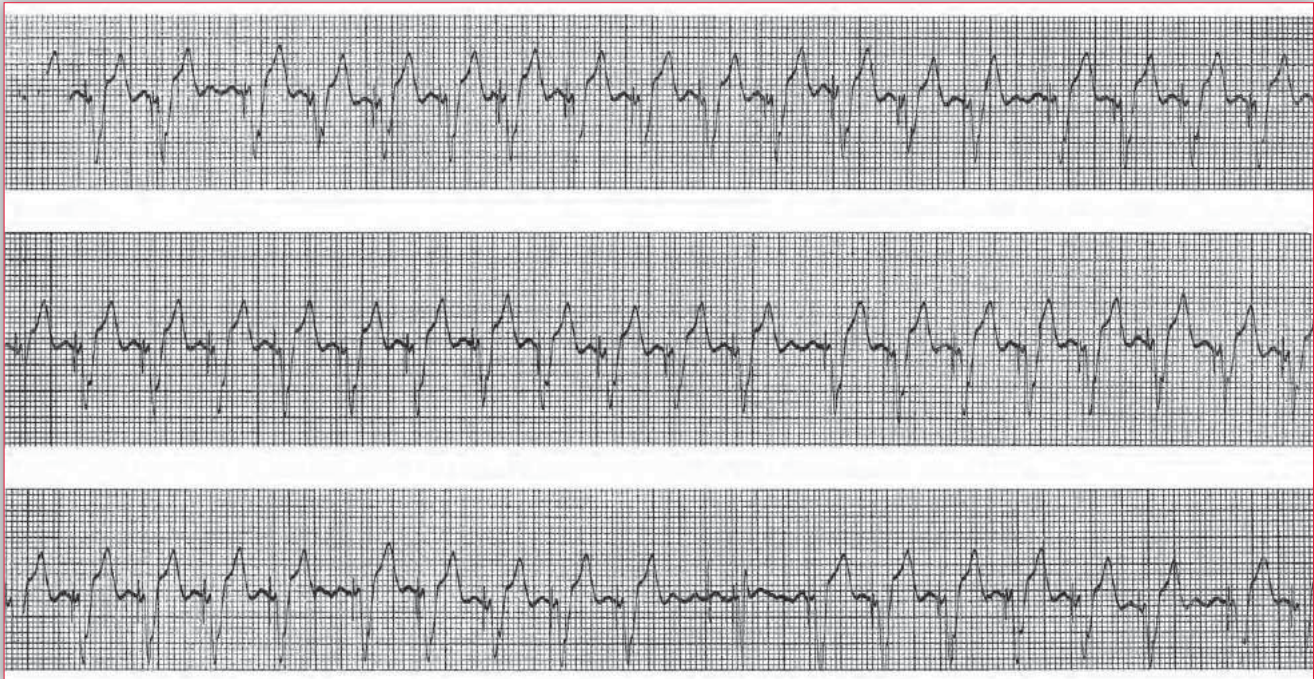
Practice Case 104

An 80-year-old woman with recent pacemaker placement for sick sinus syndrome presents with acute-onset palpitations. The following ECG is obtained.

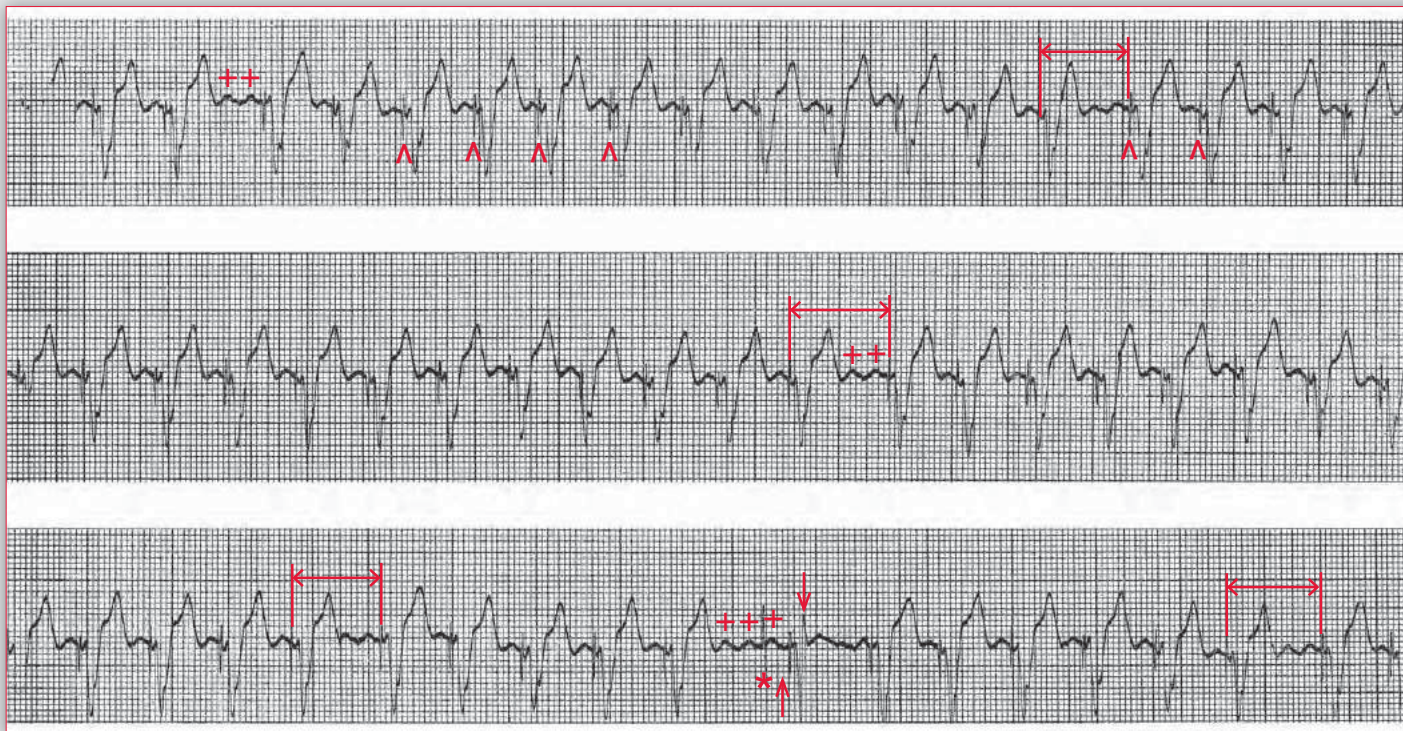
What is the rhythm abnormality?

What type of pacemaker does the patient have?

How will you acutely treat this patient's palpitations?



Podrid's Real-World ECGs



ECG 104 Analysis: Pacemaker-associated tachycardia, atrial flutter, P-wave activated ventricular pacing, AV sequential pacing, pseudofusion

ECG 104 rhythm strips show a regular wide complex tachycardia at a rate of 130 bpm, although there are some longer RR intervals seen (\leftrightarrow) that have variable durations. There is a pacemaker stimulus seen before each QRS complex (\wedge). As the ventricular pacing rate is 130 bpm, there is a dual-chamber pacemaker and the atrial lead is sensing atrial activity and pacing the ventricle; hence this is P-wave synchronous ventricular pacing or atrial sensed ventricular paced (*ie*, DDD pacemaker). During the long RR intervals, regular atrial waveforms can be seen ($+$), at a rate of 300 bpm. Hence the underlying rhythm is atrial flutter. It is likely that the pacemaker is pacing at its upper rate limit. In addition, not all of the atrial flutter waves are sensed, accounting for the longer RR intervals without ventricular pacing stimuli being seen.

The third (bottom) strip shows a longer RR interval, ending with an episode of AV sequential pacing (atrial stimulus [$*$] followed by a ventricular stimulus [\uparrow] after a delay of 0.18 sec). This represents the AV delay of the pacemaker. The ventricular stimulus does not completely capture the ventricle, as the QRS complex is narrow (\downarrow) with a morphology that is different than the paced QRS complexes. This is termed pseudofusion. The presence of the atrial pacing spike indicates that there was a longer period during which the atrial lead failed to sense the atrial flutter waves. The duration from the last paced QRS complex to the atrial stimulus represents the lower pacing limit of the pacemaker (*ie*, rate 80 bpm). Hence the wide complex tachycardia is pacemaker associated, as a result of the pacemaker tracking the atrial flutter waveforms and pacing at the upper rate limit of the pacemaker (*ie*, 130 bpm). However, there are episodes when the atrial flutter waves are not sensed by the atrial lead and if the duration of failing to sense

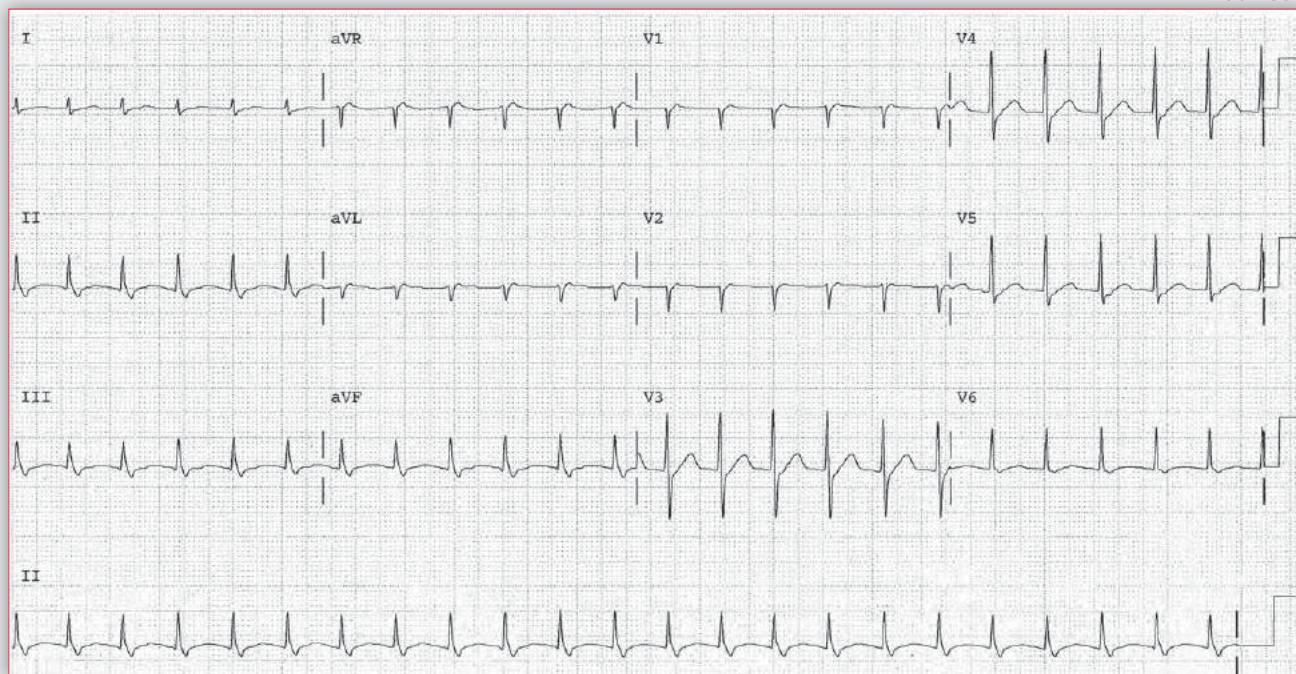
the atrial waves is long enough, there is an atrial stimulus, which will not result in atrial capture because of the rapid atrial rate. The failure to intermittently sense the atrial flutter waves results from the timing of the atrial flutter wave in relation to the ability of the atrial lead to sense the atrial impulse as well as where the atrial impulse enters the atrial myocardial to stimulate the atrium and the location of the atrial lead.

Most pacemakers have a programmable feature that results in mode switching, *ie*, if a rapid atrial rate is sensed, the pacemaker automatically reverts to a VVI mode, so as to avoid tracking of the rapid atrial rate. However, if this does not occur, a pacemaker-associated tachycardia can be treated by placing a magnet over the pacemaker. The magnet will turn off sensing (*ie*, VOO mode if a single-chamber ventricular pacemaker or DOO mode if a dual-chamber pacemaker). In this situation, since there is a DDD pacemaker, a DOO mode will result in atrial and ventricular pacing at the lower rate limit independent of the intrinsic atrial and ventricular impulses (*ie*, no sensing). Hence there will be evidence of AV sequential pacing at the lower rate limit. Atrial or ventricular capture will occur only when the atrial or ventricular myocardia are capable of being stimulated, which is dependent upon the intrinsic atrial and ventricular rates. After the magnet is placed acutely, the patient can be managed with the typical treatments for atrial flutter, such as nodal agents or antiarrhythmic drugs and/or cardioversion. Long term, a pacemaker can be reprogrammed to VVI pacing, if the patient remains in a persistent atrial arrhythmia, or mode switching if the atrial arrhythmia is paroxysmal. ■

Practice Case 105

A 21-year-old college student presents with intermittent palpitations while studying for the MCATs. Although the episodes have been brief, one prolonged episode occurred after he had several cups of coffee. In the emergency department an ECG is obtained (ECG 105A). While waiting for the emergency department physician, the arrhythmia abruptly terminates and a follow-up ECG is obtained (ECG 105B).

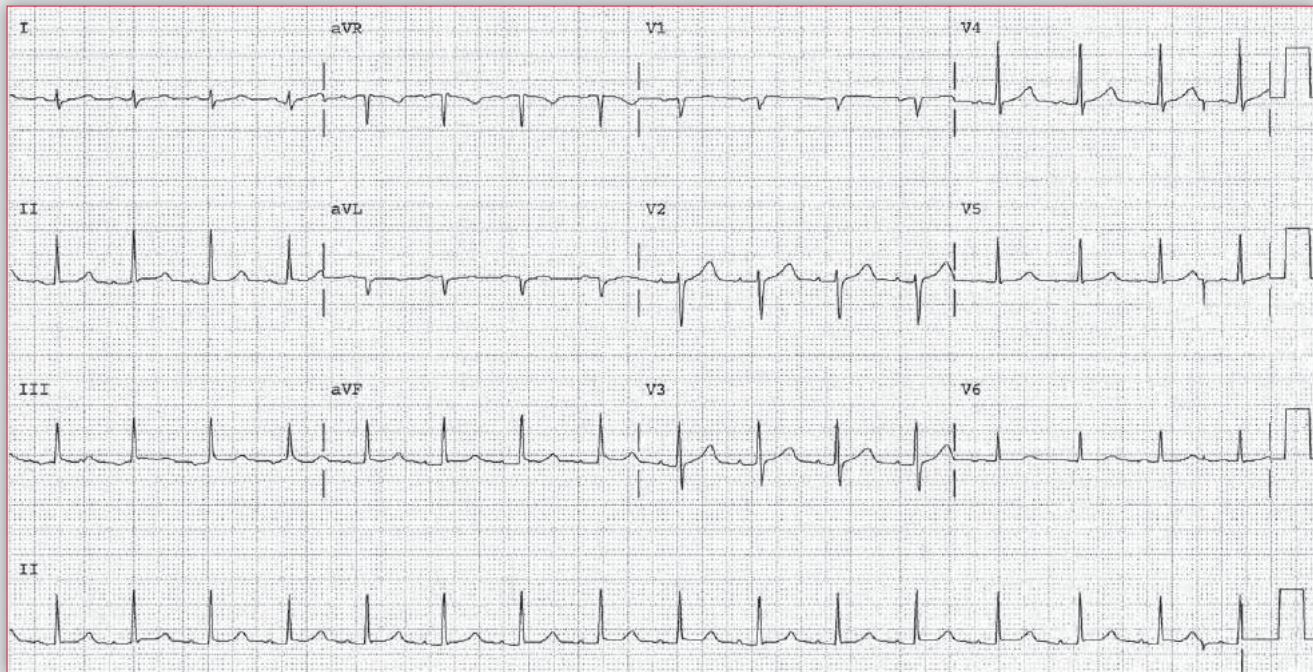
ECG 105A



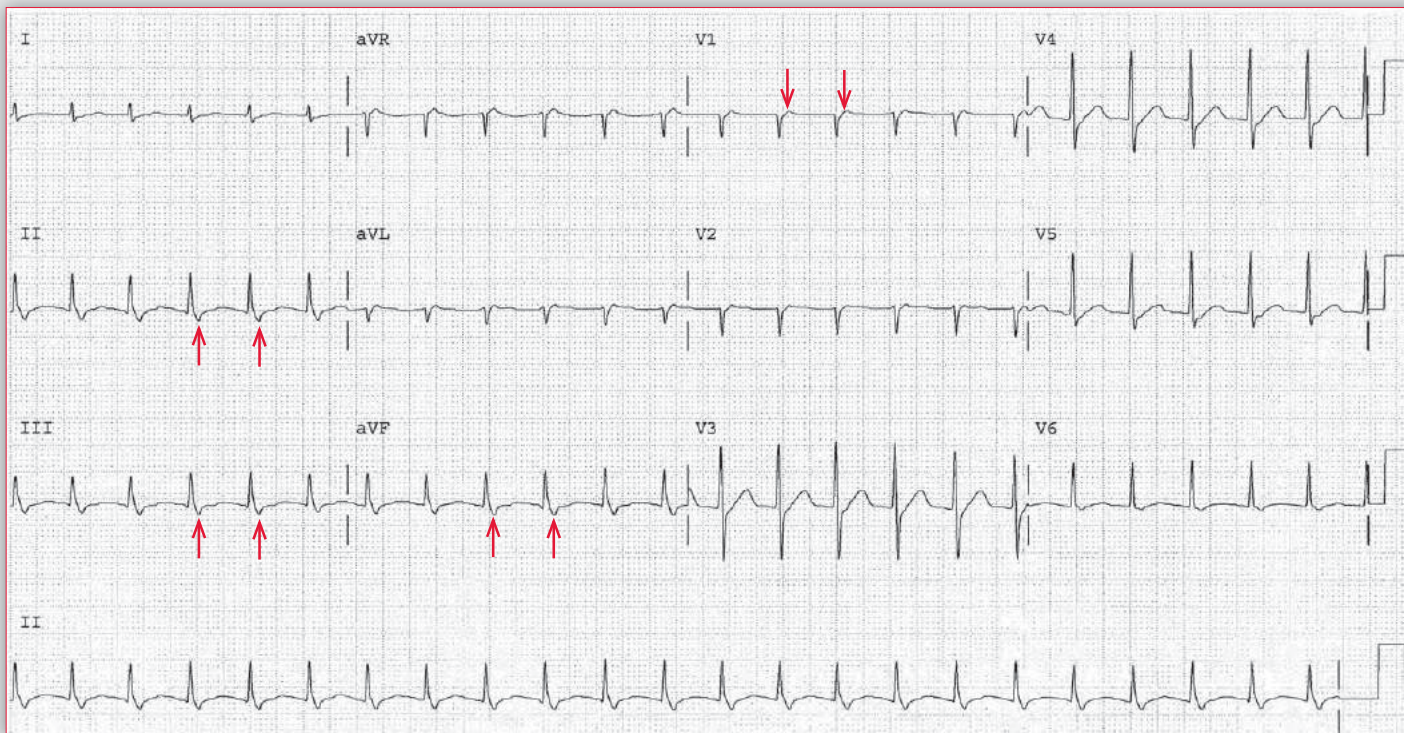
Practice Case 105

What is the rhythm abnormality?

ECG 105B



Podrid's Real-World ECGs



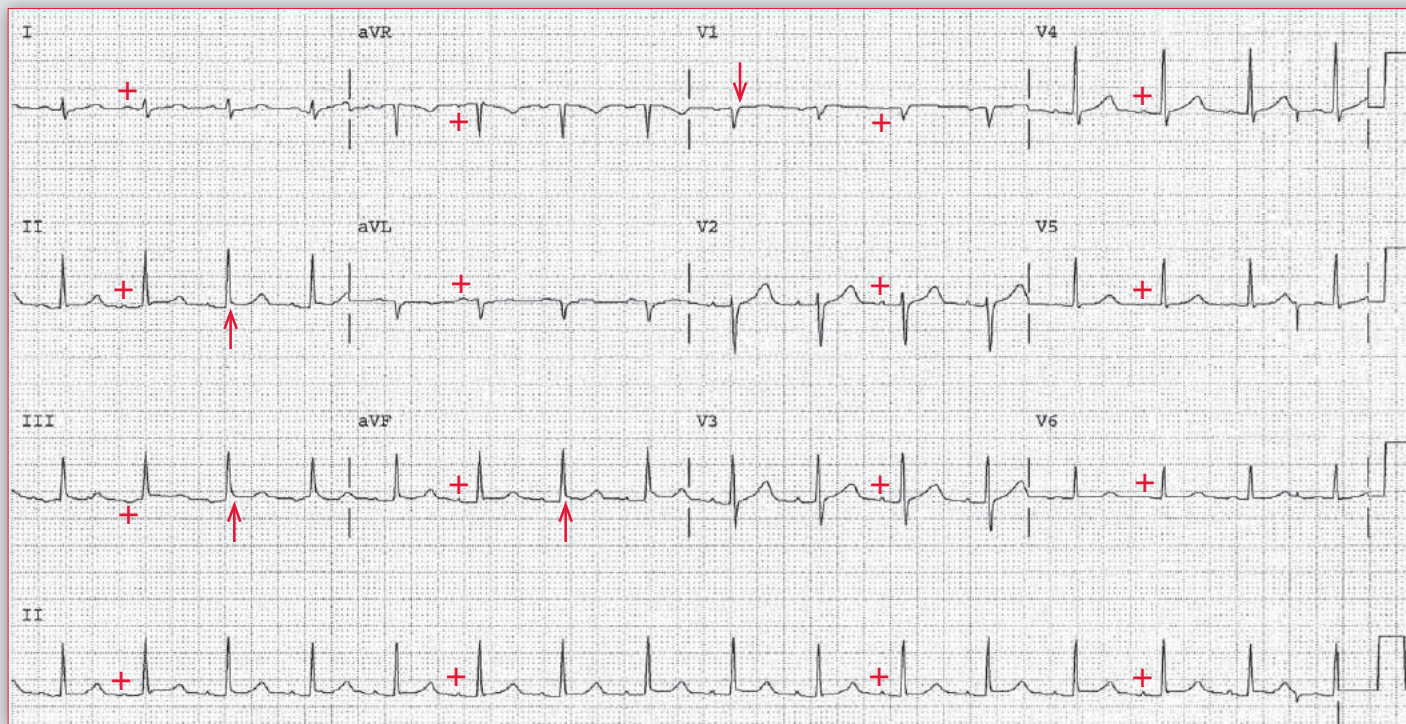
ECG 105A Analysis: Supraventricular tachycardia, short RP tachycardia, atrioventricular nodal reentrant tachycardia

ECG 105A shows a regular rhythm at a rate of 134 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/420 msec). There are no P waves seen before or after any of the QRS complexes. However, there is a positive waveform seen immediately after the QRS complex in lead V1 (\downarrow), that resembles an R but it is slightly after the QRS complex. There is also what appears to be an S wave in leads II, III, and aVF (\uparrow). This waveform likely represents a retrograde P wave, although to be certain about the etiology of this waveform,

this ECG should be compared to the baseline ECG. Hence this is a short RP tachycardia. Etiologies include sinus tachycardia (which can be eliminated as the P waves are negative in leads II and aVF), atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, atrioventricular reentrant tachycardia, or an uncommon form of a common or typical atrioventricular nodal reentrant tachycardia, known as slow-slow. In this situation, the slow pathway conducts the impulse antegradely to the ventricles, while the fast pathway that conducts the impulse retrogradely to the atrium conducts relatively slowly as a result of age-related changes or the effect of a drug.

continues

Podrid's Real-World ECGs



ECG 105B Analysis: Sinus tachycardia

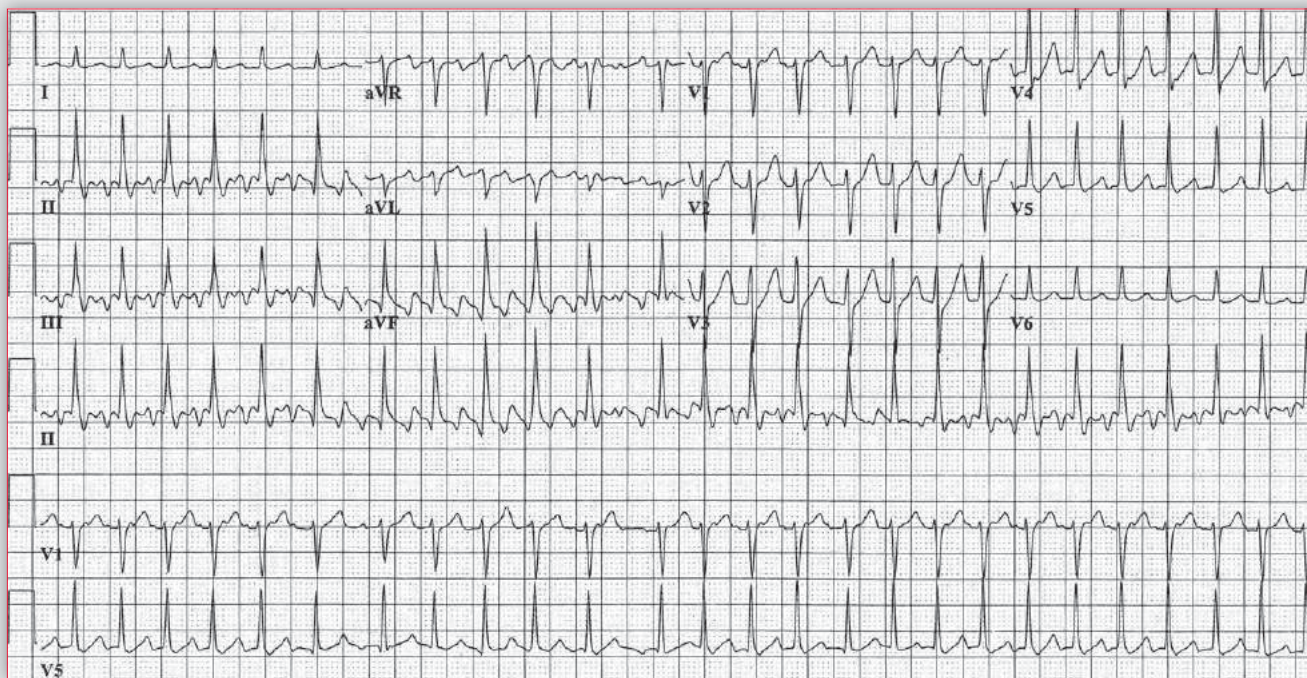
ECG 105B is from the same patient as ECG 105A. It is the patient's baseline ECG. There is a regular rhythm at a rate of 100 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The QRS complex duration and axis are the same as seen in ECG 105B. The QT/QTc intervals are the same as ECG 105A. However, the QRS complex morphology is different as the positive wave form at the end of the QRS complex in lead V1 (↓) and the S waves in leads II, III, and aVF (↑) are not present. Thus, this confirms that this waveform is a retrograde P wave that is slightly after the QRS complex. The most likely etiology is a typical atrioventricular nodal reentrant tachycardia (AVNRT) of a slow-slow type. AVNRT is the result of dual AV nodal pathways that are linked proximally and distally within the AV node. There is a fast pathway that conducts

rapidly, but has a long recovery time, *ie*, long refractory period. The slow pathway conducts slowly, but has a short refractory period or recovery time. Under normal circumstances, the fast pathway predominates. However, if there is a premature atrial impulse that arrives at the AV node before the fast pathway has recovered, the impulse will enter the slow pathway (if it has recovered), to be conducted antegradely to the ventricles. If it arrives at the distal end of the circuit at a time when the fast pathway has recovered, it will conduct through the fast pathway retrogradely to the atria. If retrograde conduction through the fast pathway is relatively slow, then atrial activation will occur slightly after ventricular activation. If the impulse arrives at the proximal portion of the circuit at a time that the slow pathway has recovered, the impulse will reenter this pathway. If this process continues, a reentrant arrhythmia will be established, *ie*, an AVNRT. ■

Practice Case 106

You are examining a patient with a history of diastolic heart failure who presents with palpitations and lightheadedness. You note that the pulse is rapid and regular, although at times it is irregular. ECG 106A is obtained and compared to ECG 106B, which is the patient's baseline ECG.

ECG 106A

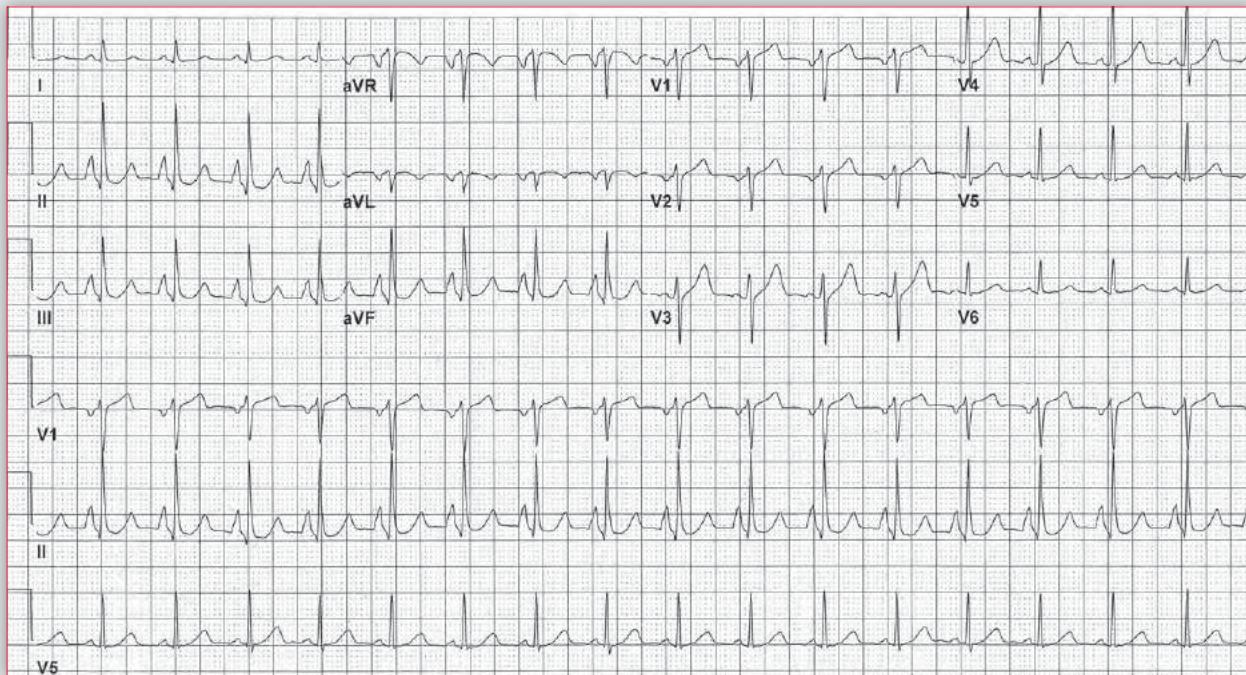


Practice Case 106

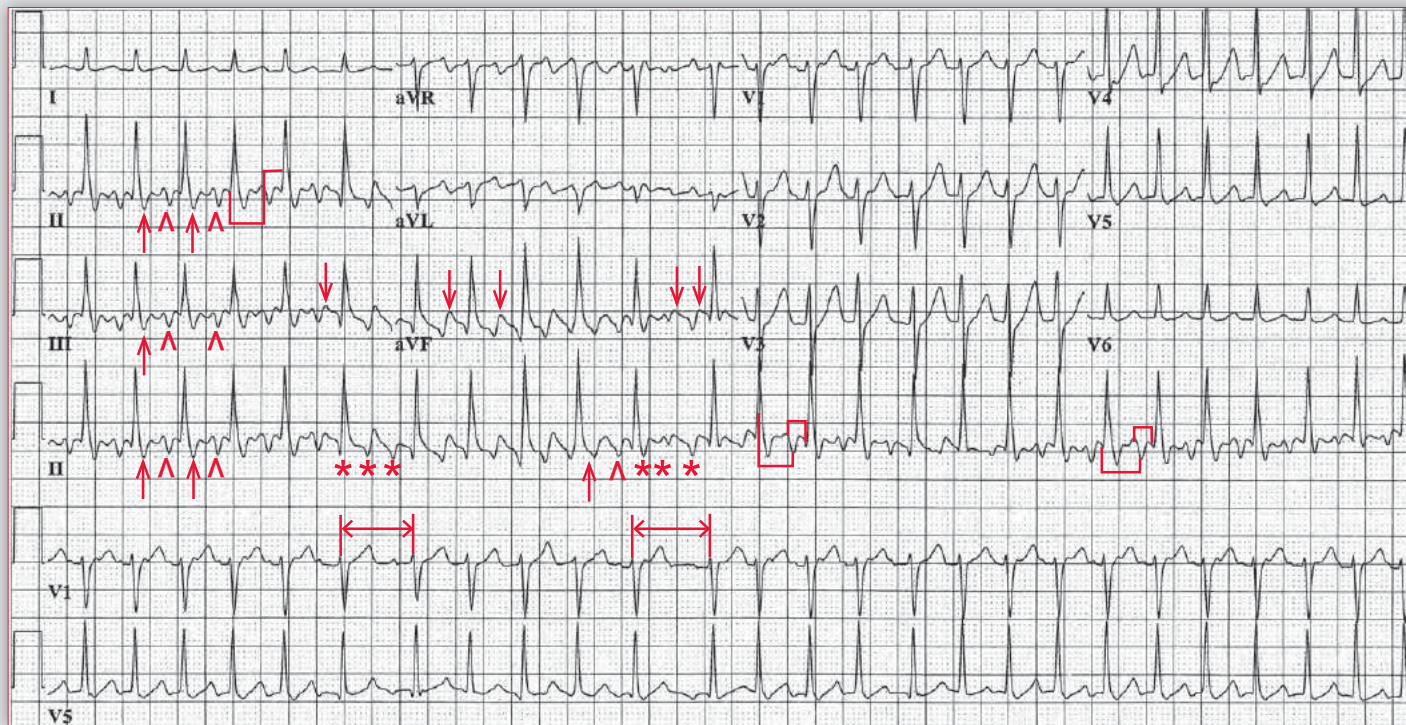
What is causing the underlying arrhythmia?

What is the cause for the irregularity in the heart rate?

ECG 106B



Podrid's Real-World ECGs



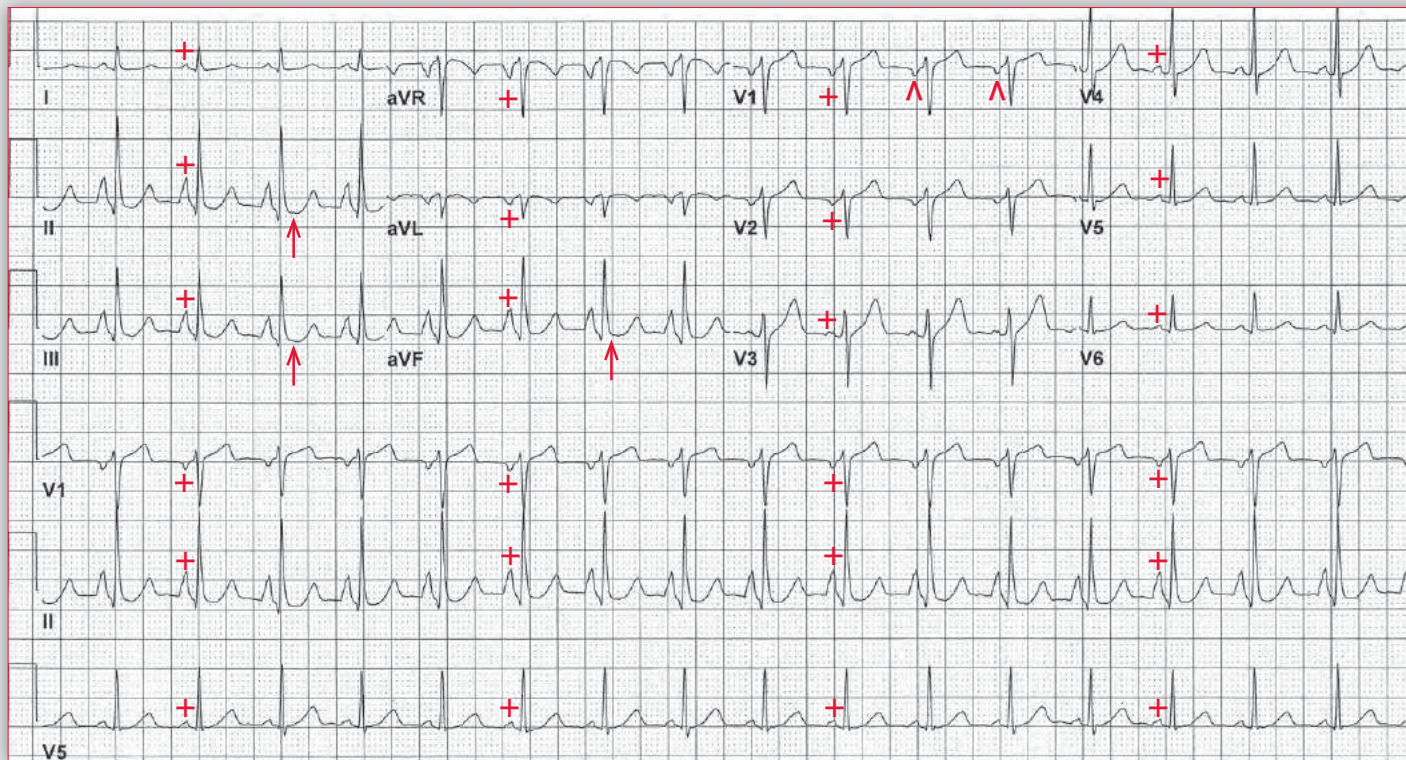
ECG 106A Analysis: Atrial flutter with primarily 2:1 AV block, intermittent 3:1 AV block, antegrade concealed conduction

ECG 106A shows a regular rhythm, although two long RR intervals are seen (\leftrightarrow). The rate is 160 bpm. The QRS complexes have a normal duration (0.08 sec) and morphology. The axis is normal between 0 and +90 (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (260/425 msec). Although there are no obvious P waves seen before or after any QRS complex, there are negative waveforms seen in leads II, III, and aVF between each QRS complex (\wedge). As the RP interval (\sqcup) is longer than the PR (\sqcap), this would be considered a long RP tachycardia. However, with careful inspection it can be seen that there is a similar waveform at the end of the QRS complex in leads II, III, and aVF (\uparrow), which looks like an S wave. However, there is a stable interval between this waveform and the negative waveform, at

a rate of 320 bpm. In addition, multiple atrial waveforms can be seen during the long RR intervals (*), at the same rate (320 bpm) and they maintain the constant interval. Hence this is atrial flutter with primarily 2:1 AV block. The two long RR intervals are the result of transient 3:1 AV block. Just prior to and after the longer RR intervals, there is variability in the interval between the flutter waves and the QRS complexes (\downarrow), which is a result of antegrade concealed conduction. Some atrial impulses are conducted, others are completely blocked, and some partially penetrate the AV node and partially depolarize it (concealed within the AV node). A subsequent impulse may be conducted through the AV node, but with a longer conduction time.

continues

Podrid's Real-World ECGs



ECG 106B Analysis: Normal sinus rhythm, right atrial hypertrophy, left atrial hypertrophy (biatrial hypertrophy)

ECG 106B is from the same patient as ECG 106A. There is a regular rhythm at a rate of 100 bpm. The QRS complex duration and axis are the same as in ECG 106A. There is a P wave before each QRS complex (+) with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V6; hence this is a sinus rhythm. However, the P waves are tall, narrow, and peaked in leads II, III, and aVF, characteristic of right atrial hypertrophy, while they are only negative

in leads V1–V2 (^), which is characteristic of left atrial hypertrophy. Hence there is biatrial hypertrophy. Note that there is no S wave seen in leads II, III, and aVF (↑), establishing that this was indeed the second flutter wave. Otherwise, the QRS complex morphology is the same as seen in ECG 106A. The QT/QTc intervals are normal (320/410 msec). ■

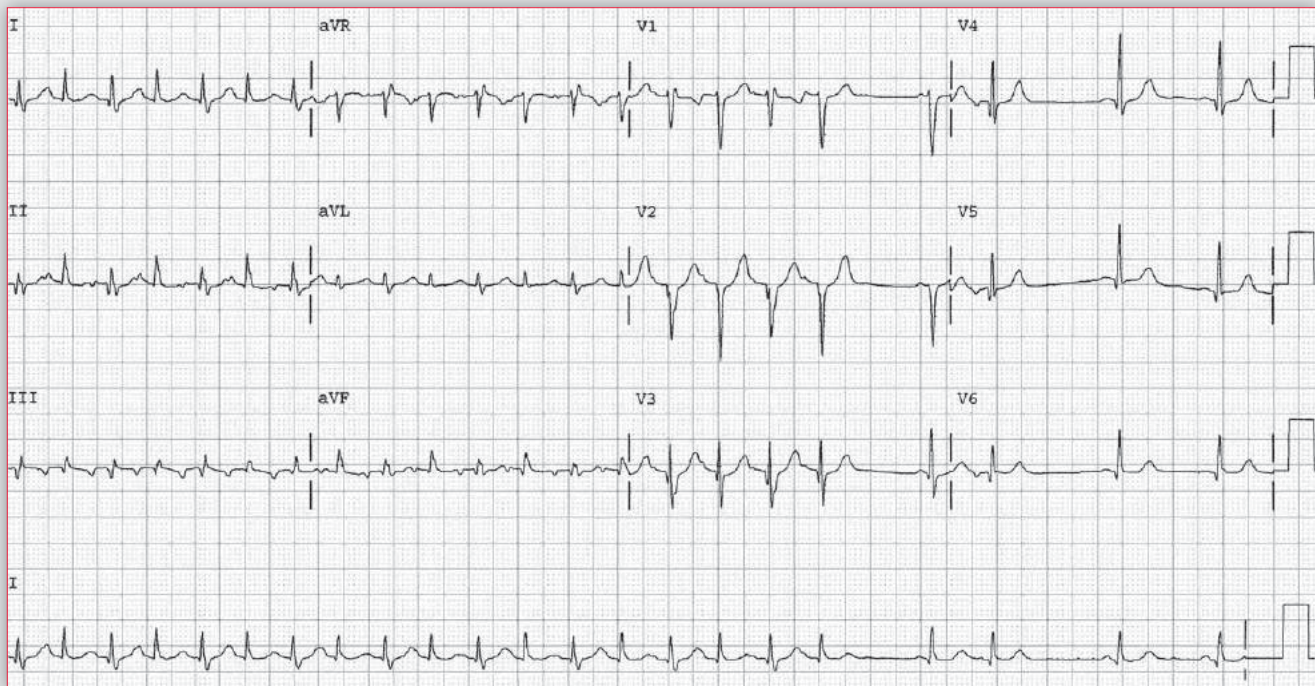
Notes

Practice Case 107

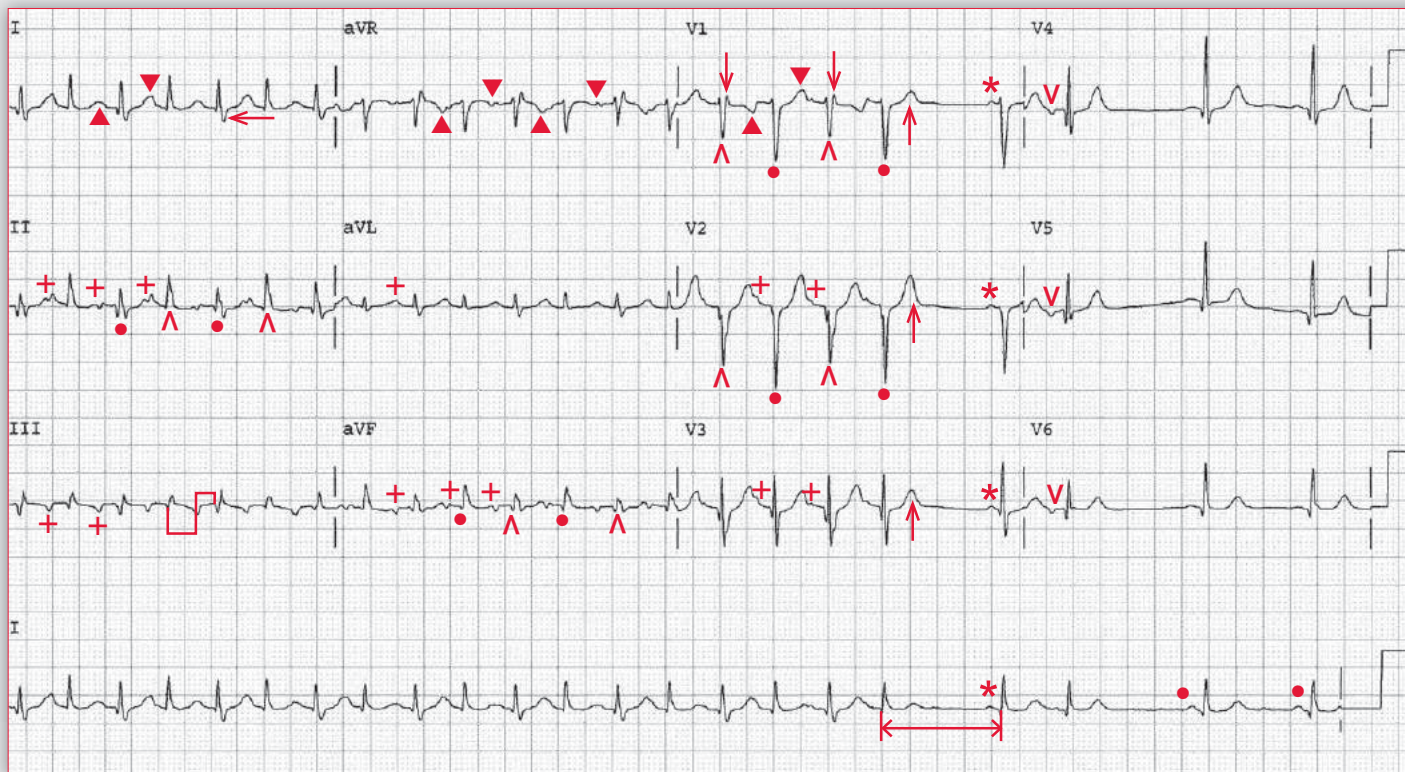
A 34-year-old man with HIV presents with dyspnea, fatigue, a productive cough and intermittent palpitations over the past 3 days. On examination, he is cachectic, tachycardic, and hypotensive with a blood pressure of 90/60. Chest x-ray confirms pneumonia and there is mild cardiomegaly. The following ECG is obtained. Based on the ECG, a stat echocardiogram is ordered, which showed a slightly dilated left ventricle with an LVEF of 50% but was otherwise normal.

What does the ECG show that is of concern?

What is the underlying rhythm?



Podrid's Real-World ECGs



ECG 107 Analysis: Atrial tachycardia, long RP tachycardia, electrical (QRS) and T-wave alternans

ECG 109 showed the initial rhythm (first 18 QRS complexes) to be a regular rhythm at a rate of 160 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). Also noted are beat-to-beat changes in the QRS complex amplitude (▲,●), known as electrical or QRS alternans. In addition, there are beat-to-beat changes in T-wave amplitude (▼,▲), known as T-wave alternans. As a result of the electrical alternans, every other QRS complex has a small R' in lead V1 (↓) and S wave in leads I and II (←), reflecting a beat-to-beat evidence of a right ventricular conduction abnormality. The QT/QTc intervals are slightly prolonged (280/460 msec).

There are P waves (+) noted in between each QRS complex, most obviously in leads I, II, III, aVF, and V1–V3. The P waves are negative in leads II, III, and aVF. The RP interval (⊐) is 0.24 second, and the PR interval (⊐) is 0.16 second. This is a long RP tachycardia. Etiologies include sinus tachycardia (which is excluded as the P waves are negative in leads II and aVF), atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, atypical atrioventricular nodal reentrant tachycardia (known as fast-slow, *ie*, the antegrade activation of the ventricles is via the fast pathway while retrograde activation of the atria is via the slow pathway) or atrioventricular reentrant tachycardia (associated with an accessory pathway that may be overt or concealed).

Seen is the acute termination of the tachycardia (↔) and there is no P wave present after the last QRS complex prior to termination (†). Thus, the arrhythmia terminates with the absence of atrial activity, which is characteristic of atrial arrhythmias; this is most likely an atrial tachycardia. Reentrant rhythms involving the node such as AVNRT and

AVRT typically terminate with a final retrograde P wave (which is non-conducted). The first QRS complex after the arrhythmia terminates (complex 19) has a P wave (*) before it with a PR interval of 0.16 second; this is a sinus complex. The QRS complex has the same duration and morphology as those during the atrial tachycardia. Following this sinus complex, there is a premature atrial complex with a negative P wave (v). Thereafter, there are two additional sinus complexes, with the same P wave (●) as seen with the first QRS complex after the pause. The sinus rate is 76 bpm with a constant PR interval of 0.16 second. It should be noted that there is no evidence of electrical alternans during normal sinus rhythm.

Electrical or QRS alternans has most often been attributed to a large pericardial effusion and tamponade. This was the concern in this patient given the clinical presentation. In this situation, the alternans is due to the swinging of the heart in a fluid-filled sac (pendulum effect). Infrequently, P-wave alternans may also be seen with tamponade. There are other causes for electrical alternans that include an acute myocardial infarction, a severe dilated cardiomyopathy, decompensated heart failure, and any supraventricular tachycardia (atrial tachycardia, atrial flutter, atrioventricular nodal reentrant tachycardia, or atrioventricular reentrant tachycardia). Although it has been stated that electrical alternans is a feature of an atrioventricular reentrant tachycardia (due to an accessory pathway), this is likely because these arrhythmias tend to have faster heart rates than other etiologies for supraventricular tachycardia. The electrical alternans seen with these other conditions is the result of beat-to-beat change in calcium fluxes into the myocardium. Interestingly, P-wave alternans is not seen with these conditions. ■

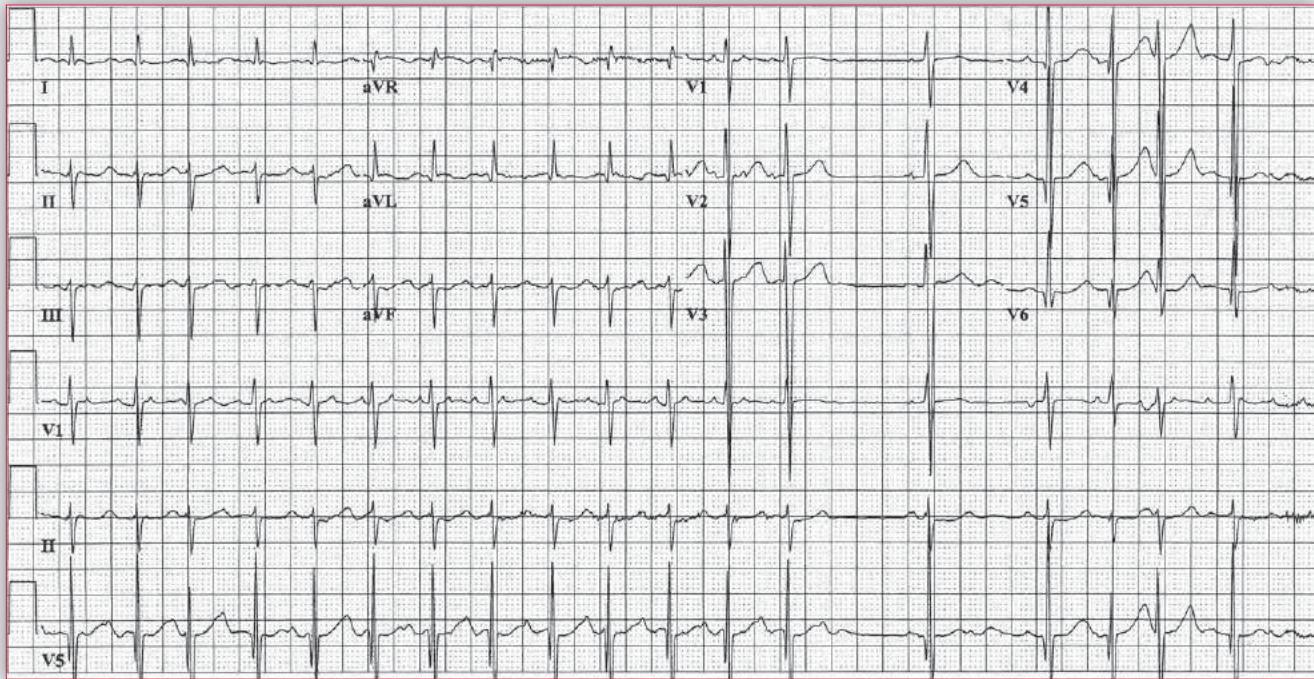
Notes

Practice Case 108

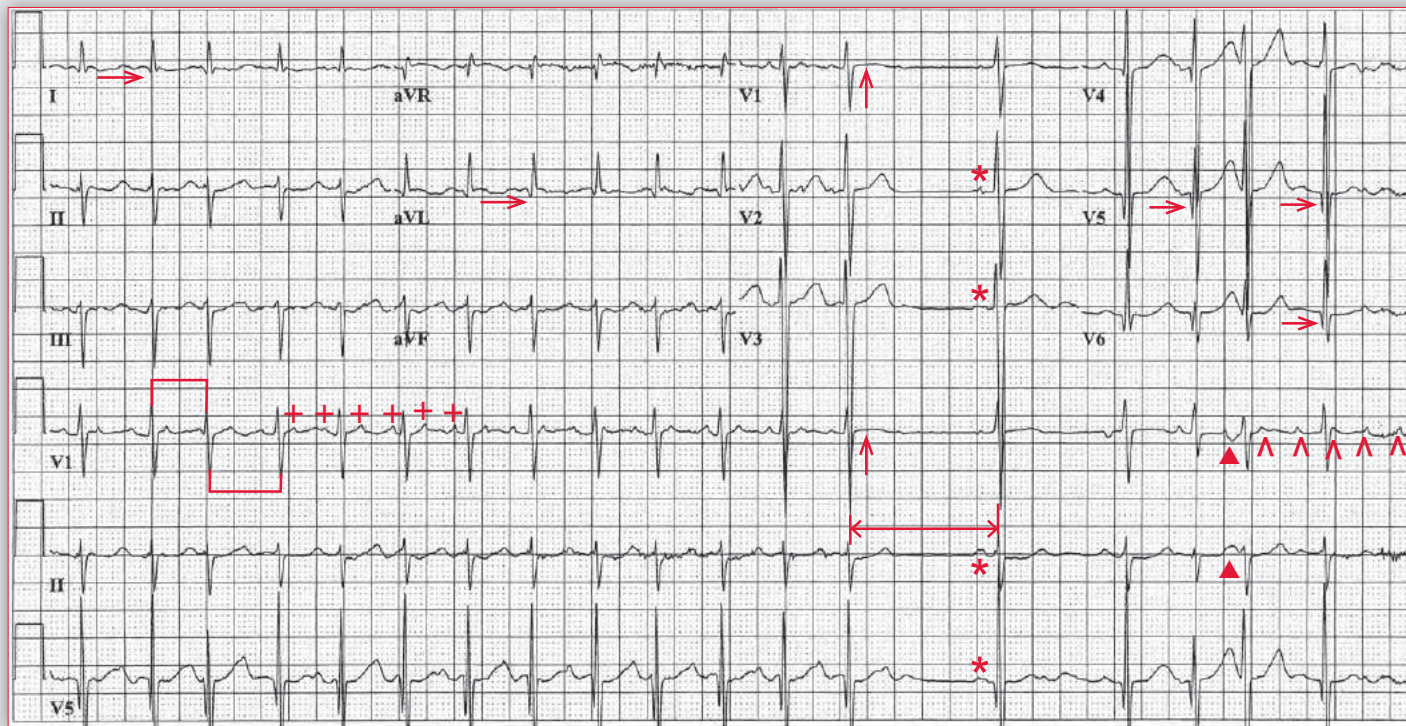
A 62-year-old woman with diabetes but no known coronary artery disease presents with 2 days of progressive dyspnea, orthopnea, lower extremity swelling, and palpitations. She also reports that one week ago, she suffered from reflux-like symptoms for an entire day, but that these symptoms resolved on their own. She did not seek medical attention until today. The following ECG is obtained.

What is the rhythm abnormality?

What most likely precipitated her clinical symptoms?



Podrid's Real-World ECGs



ECG 108 Analysis: Intermittent atrial flutter with 2:1 AV block, left anterior fascicular block, old lateral wall myocardial infarction, antegrade concealed conduction

ECG 108 showed the initial rhythm (first 13 complexes) is primarily regular, with one slightly shorter RR interval (□) (complexes 2–3) and one slightly longer RR interval (□) (complexes 3–4). The rate is 134 bpm. There are two P waves (+) seen within each RR interval, most obvious in lead V1. The P waves are regular at a rate of 280 bpm. This is atrial flutter with 2:1 AV block. The slight irregularity of the two RR intervals is the result of antegrade concealed conduction that results from occasional atrial impulses that penetrate the AV node, but fail to conduct completely through the node (concealed within the AV node). However, they partially depolarize the AV node, altering its refractoriness such that the next atrial impulse conducts through the node at a slightly slower rate.

There is an abrupt slowing of heart rate due to termination of the atrial flutter (↔). The complex after the pause is likely sinus (*). It should be noted that the atrial flutter terminates with the absence of atrial activity (†), which is the way atrial arrhythmias terminate, *ie*, the reentrant mechanism or ectopic focus within the atrial myocardium stops and hence there is no atrial waveform seen. The last portion of the ECG shows recurrence of the atrial flutter (with an atrial rate identical to the initial flutter rate) (^) that appears to be initiated by a premature atrial complex (▲).

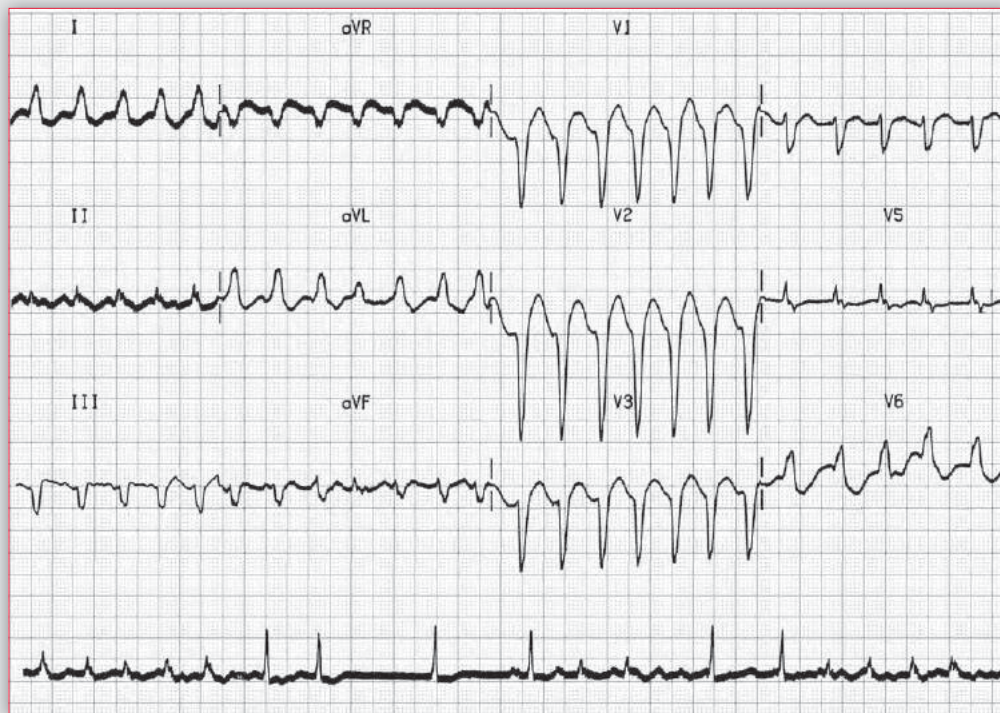
The QRS complex duration is normal and there is an extreme left axis between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF with a r/S morphology). This is a left anterior fascicular block. In addition, there are Q waves (→) in leads I, aVL, and V5–V6, suggesting an old lateral/anterolateral wall myocardial infarction.

The clinical scenario suggests that the patient suffered from a lateral wall myocardial infarction one week ago and has now developed progressive heart failure, possibly related to left ventricular dysfunction from the myocardial infarction (although a mechanical complication must also be considered). In addition, the atrial flutter is also likely contributing to her heart failure symptoms, although it is not certain if the atrial flutter is contributing to or the cause of the heart failure or is the result of heart failure. Cardiac troponins should be checked, as they may still be elevated at 7 days post-MI. In addition, an echocardiogram should be obtained to determine LV function and to assess for a mechanical complication. Urgent cardiac catheterization is not indicated, as the patient has exceeded the time frame for benefit with revascularization (*ie*, > 12 to 24 hours). One can, however, perform cardiac catheterization with revascularization if there is evidence for significant residual ischemia (*ie*, on a stress test). ■

Practice Case 109

A 57-year-old woman calls 911 for distressing intermittent palpitations. On arrival to the emergency department, she appears distressed. Her vital signs are normal at first, but she manifests an intermittent tachycardia via telemetry monitoring. Other than the sensation of a “racing heart” and “panic,” she denies associated symptomatology. Consecutive tracings are obtained (ECG 109A and ECG 109B).

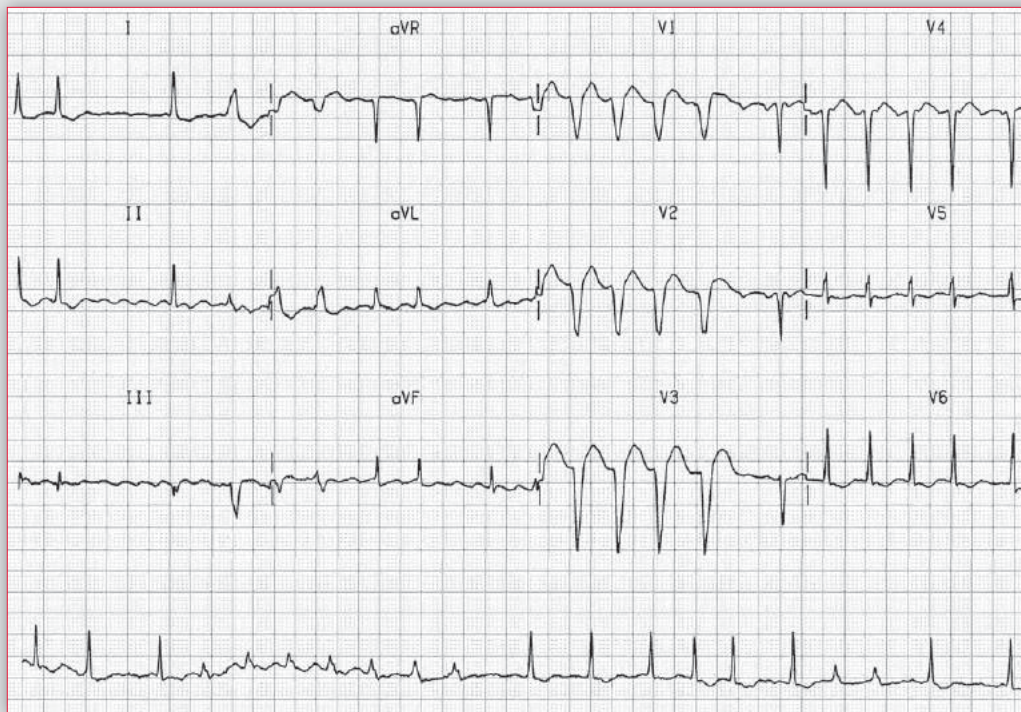
ECG 109A



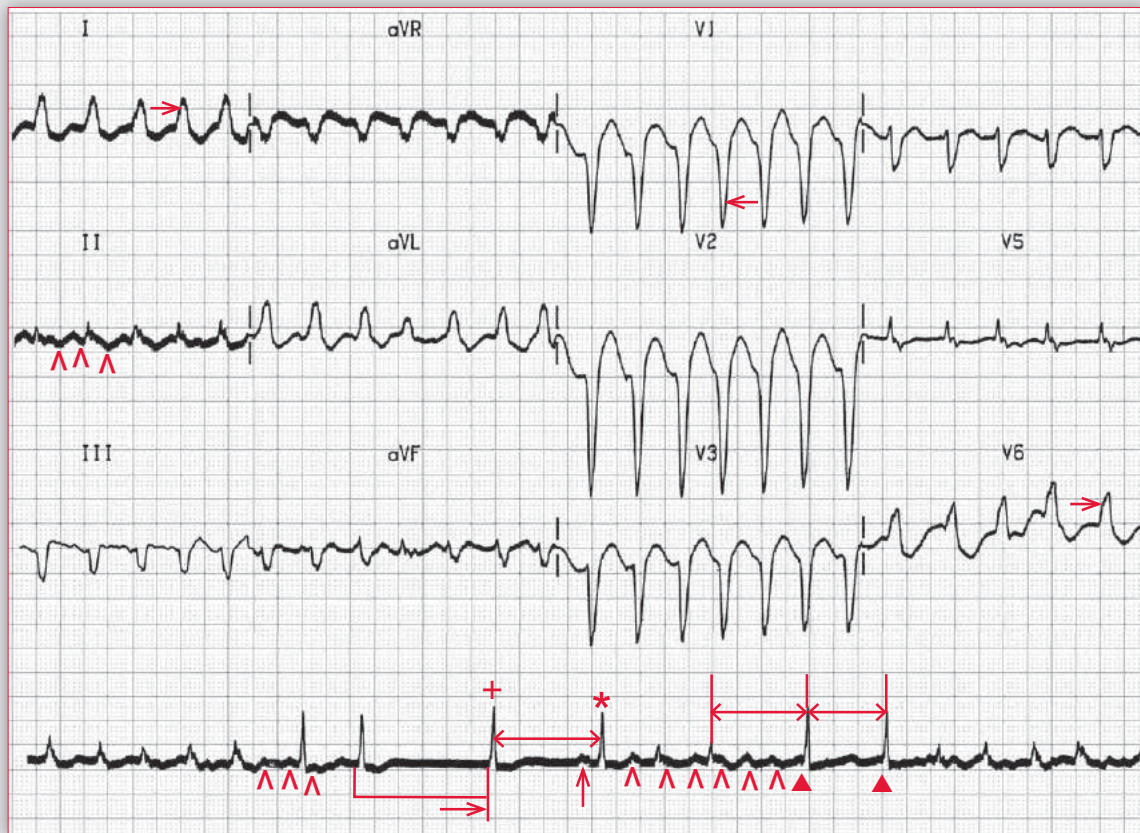
Practice Case 109

What abnormalities are noted, and what diagnosis is suggested?

ECG 109B



Podrid's Real-World ECGs



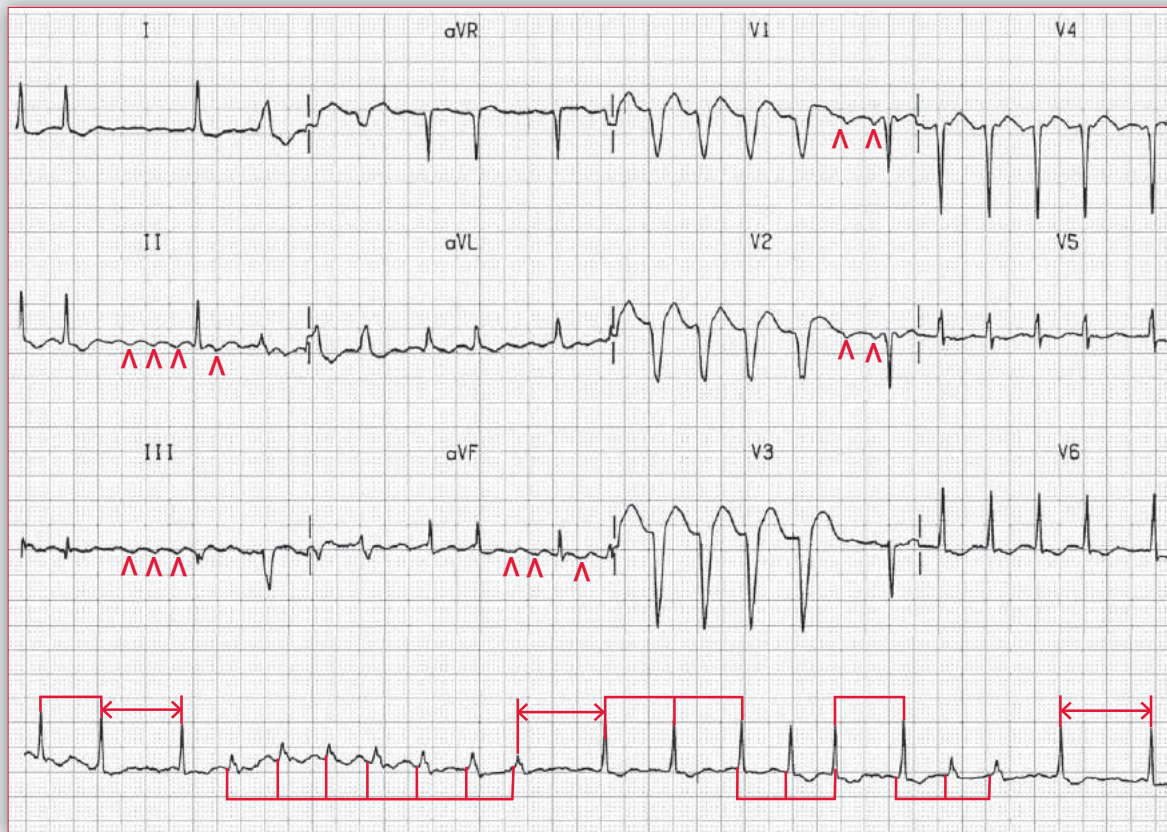
ECG 109A Analysis: Paroxysmal atrial flutter with 2:1 AV conduction, rate-related left bundle branch block morphology, junctional escape complex

ECG 109A shows a regular rhythm at a rate of 154 bpm. Note that the rhythm strip is not simultaneous with the 12-lead ECG. There are no obvious P wave seen; however, intermittently there appears to be regular atrial activity (^), especially apparent in lead II and in the rhythm strip. The atrial rate is approximately 300 bpm, consistent with atrial flutter and based on the ventricular rate this is 2:1 AV conduction. The QRS complexes have a prolonged duration (0.14 sec), and there is a left bundle branch block pattern (tall, broad R wave in leads I and V6 [→] and a QS complex in lead V1 [←]). The QT/QTc intervals are prolonged (340/500 msec) but are normal QRS complex duration is considered (300/450 msec).

In the rhythm strip there is a pause (□), during which there is no evidence for any atrial activity. The QRS complex after the pause (+) is narrow (duration 0.08), and it does not have left bundle branch block morphology. As there is no atrial activity seen before this QRS complex, it is a junctional escape complex. Following this, there is second narrow QRS complex (*), and there is a P wave before this complex (†) with a PR interval of 0.18 second. Hence this is a sinus complex. After this sinus complex there is an episode of atrial flutter with varying block. However, it can be seen that when the RR interval is long (↔) (greater degree of AV block), the QRS complex is narrow (▲) and does not have a left bundle branch block morphology. Hence there is a rate-related left bundle branch block present.

continues

Podrid's Real-World ECGs



ECG 109B Analysis: Atrial flutter with variable AV, rate-related left bundle branch block

ECG 109B is from the same patient as ECG 109A. As before, the rhythm strip is not simultaneous with the 12-lead ECG. The rhythm is irregular, but there is a pattern, *ie*, all of the long RR intervals (\leftrightarrow), all the short RR intervals (\sqcup), and all the intermediate intervals (\sqcap) are the same. Thus, the rhythm is said to be regularly irregular and the average rate is about 80 bpm. There is evidence of regular atrial activity seen at a rate of 300 bpm (\wedge), especially in leads II, III, aVF, and

V1–V2. Therefore, the rhythm is atrial flutter with varying AV block. Noted are narrow QRS complexes, which have the same width and morphology as those in ECG 109A. There are also wide QRS complexes, which have a left bundle branch block morphology and these complexes are the same as the wide QRS complexes seen in ECG 109A. The wide QRS complexes occur whenever the RR interval is short; hence this is a rate-related left bundle branch block. ■

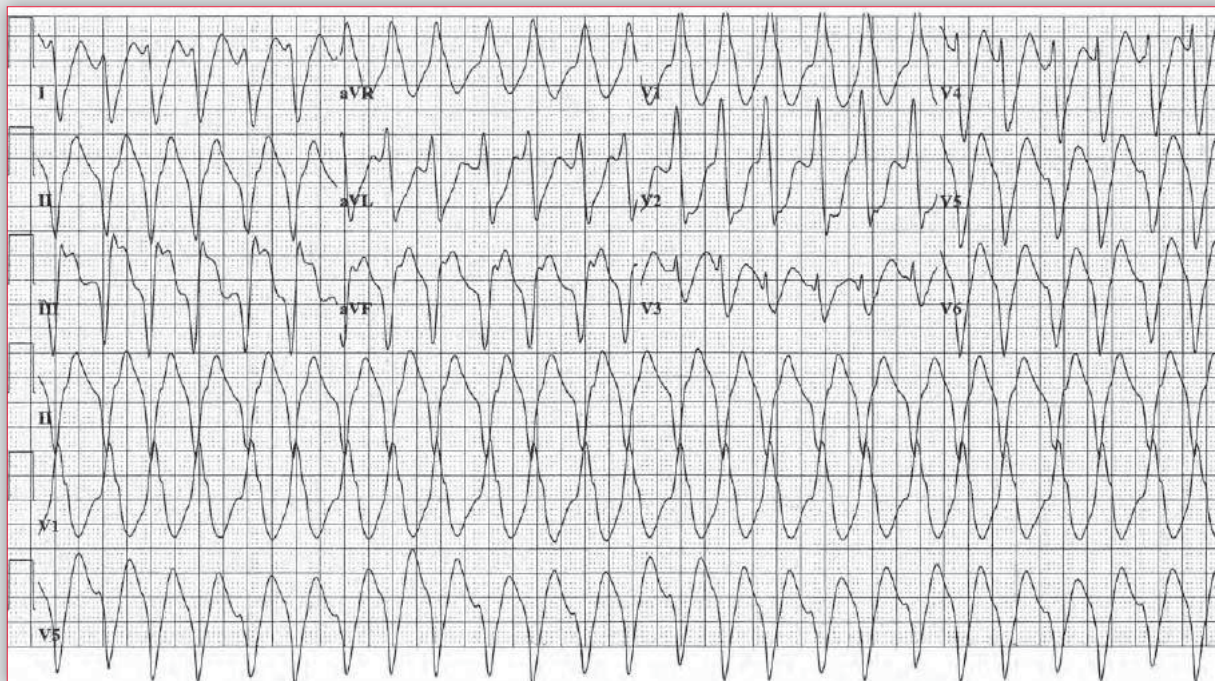
Practice Case 110

A 75-year-old man with hypertension presents to the emergency department with complaints of retrosternal chest discomfort that began the night before presentation (ie, about 8 hours). While the ECG leads are

being placed, he acutely becomes unresponsive and the 12-lead ECG is obtained (ECG 110A). What is the arrhythmia?

The patient spontaneously recovers after 1 minute and a second ECG is obtained (ECG 110B).

ECG 110A

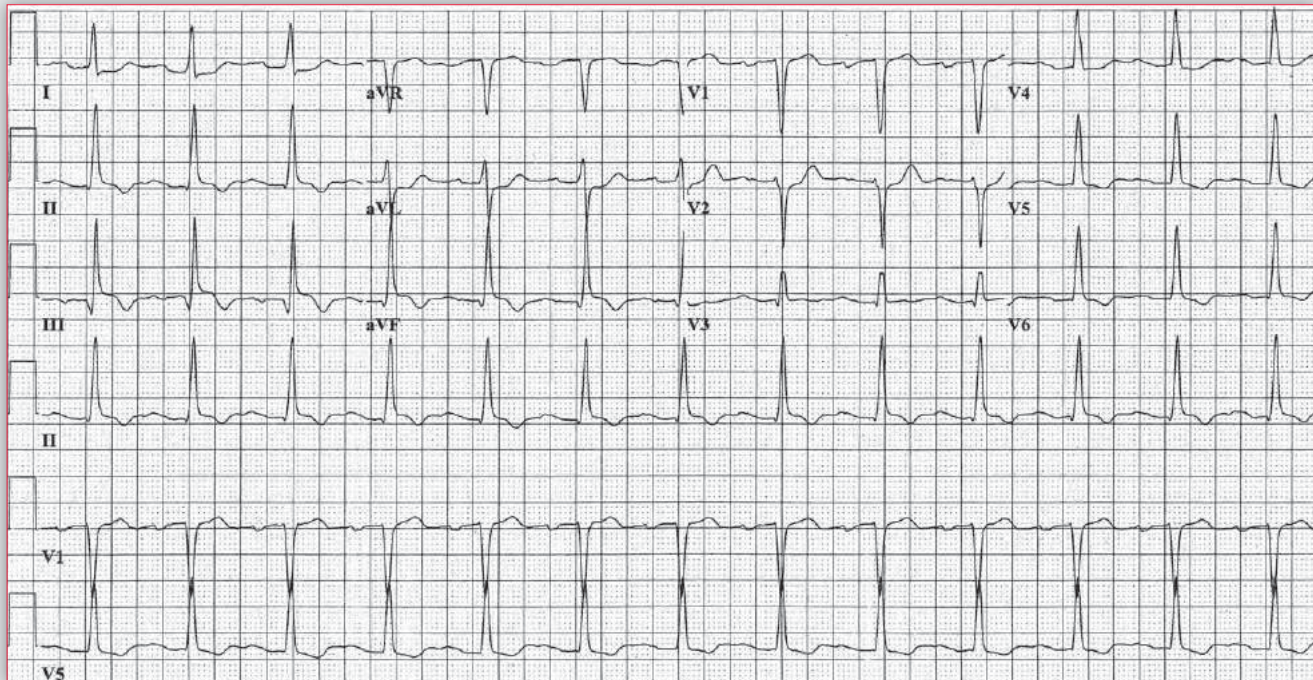


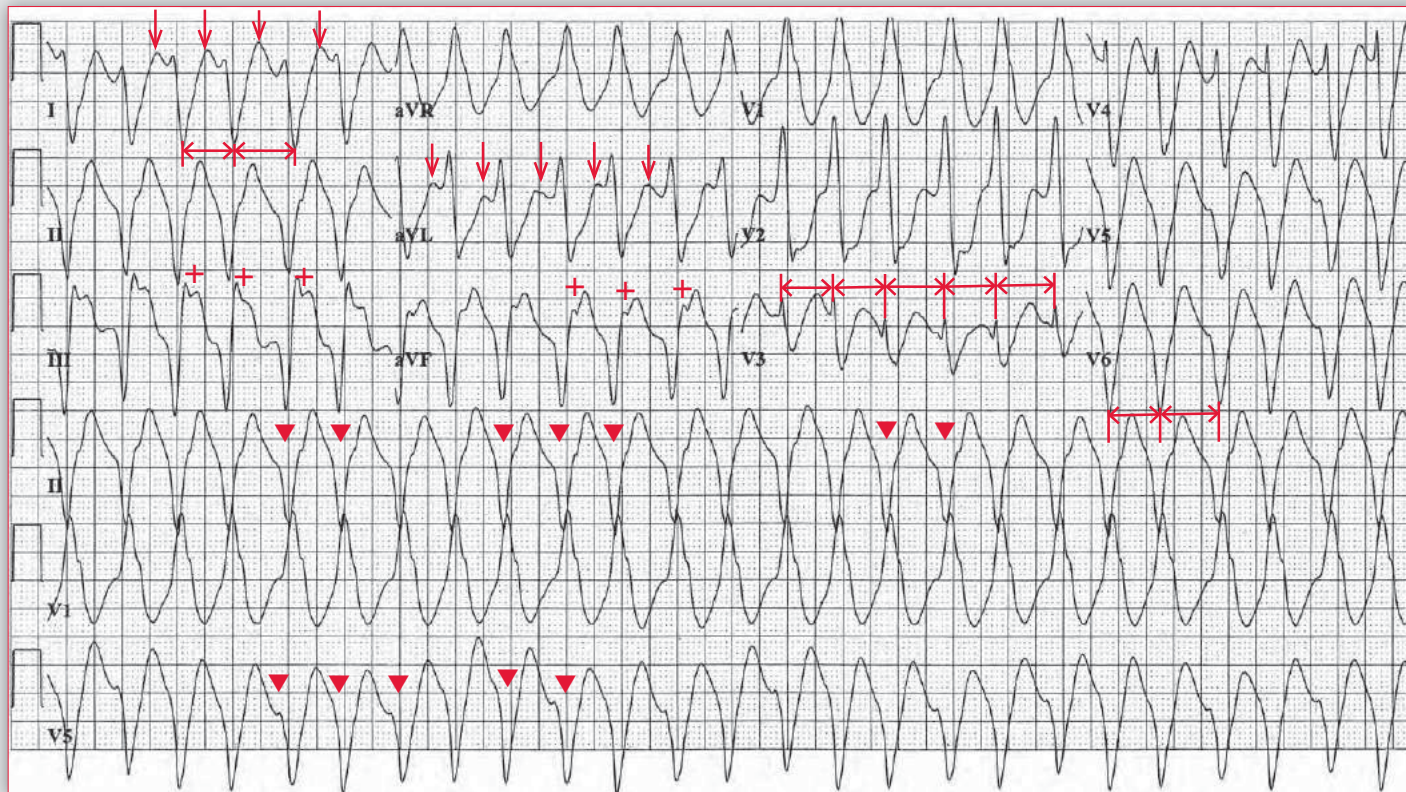
What is the etiology for the arrhythmia?

What is the cardiac diagnosis?

What is your next step in management?

ECG 110B





ECG 110A Analysis: Ventricular tachycardia

ECG 110A shows a slightly irregular rhythm (\leftrightarrow) at a rate of 152 bpm. The QRS complex duration is increased (0.18 sec). The morphology is not typical for a right or a left bundle branch block. The axis is indeterminate between -90° and $+/-180^\circ$ (negative QRS complex in leads I and aVF). There are no P waves seen before or after any QRS complex. However, there are subtle differences of QRS morphology (\blacktriangledown) as well as in the ST-T waves (\downarrow). These slight irregularities in the ST-T waves and QRS complex morphology (due to slight differences in depolarization and repolarization sequences and also possibly superimposed P waves), the indeterminate axis, and the QRS width > 0.16 second are all characteristics of ventricular tachycardia. The slight irregularity of the rhythm is often observed with ventricular tachycardia as well. Also noted is ST-segment elevation in leads III and aVF (5–8 mm). This is suggestive of an acute inferior wall myocardial infarction. However, abnormalities of the left ventricle, which include acute or chronic myocardial infarction, ischemia, left ventricular hypertrophy, pericarditis, etc., cannot be reliably diagnosed whenever left ventricular myocardial activation is not via the normal His-Purkinje system, but rather results

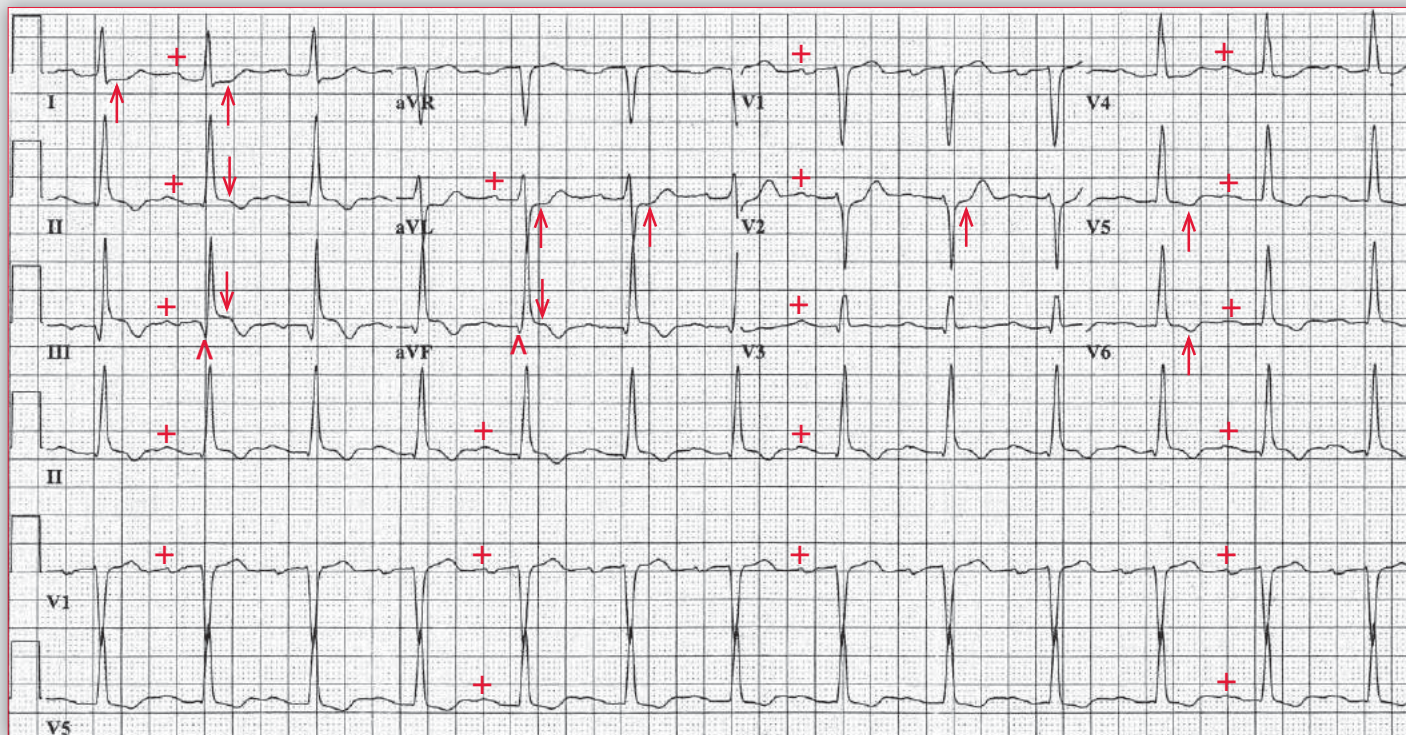
from direct myocardial activation directly through the ventricular myocardium. This includes a ventricular complex, a ventricular paced complex, a left bundle branch block, or Wolff-Parkinson-White. However, there are published criteria for diagnosing an acute myocardial infarction in the presence of a left bundle branch block or a right ventricular paced rhythm (Sgarbossa criteria), and these can also be applied to the other situations in which there is direct myocardial activation. These criteria include:

1. ST elevation > 1 mm in leads with a positive QRS complex (concordance in ST deviation)
2. ST depression > 1 mm in V1–V3 (concordance in ST deviation)
3. ST elevation > 5 mm in leads with a negative QRS complex (inappropriate discordance in ST deviation)

Therefore, the ST-segment elevation in leads III and aVF is consistent with an acute inferior wall myocardial infarction, which is also suggested by the clinical history.

continues

Podrid's Real-World ECGs



ECG 110B Analysis: Sinus rhythm with first-degree AV block,
acute inferior wall myocardial infarction

ECG 110B is the baseline ECG from the patient in ECG 110A. There is a regular rhythm at a rate of 78 bpm. There is a P wave before each QRS complex (+) with a stable but prolonged PR interval (0.28 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm with a first-degree AV block. The QRS complex duration is normal (0.10 sec). The axis is normal between 0° and +90° (positive QRS complex in lead I and aVF). There are Q waves in leads III and aVF (^), diagnostic of an inferior wall myocardial infarction. There is also ST-segment elevation in leads II, III, and aVF (↓) that may represent an acute myocardial infarction with evolutionary ST-segment changes or a chronic inferior wall myocardial infarction with an aneurysm. In addition, there are ST-segment changes (depressions) in leads I, aVL, V2, and V5–V6 (↑). The QT/QTc intervals are normal (380/430 msec).

Importantly, the ST-segment elevation in the inferior leads is also seen with the ventricular tachycardia. Given the clinical scenario, the patient is having an acute inferior MI and should proceed to revascularization emergently.

The occurrence of a sustained ventricular tachycardia in the setting of an acute myocardial infarction (*ie*, within the first 48 hours) is seen in about 4% to 5% of patients. Initial studies found that ventricular tachycardia (or ventricular fibrillation) occurring earlier after an acute myocardial infarction was not predictive of an increased mortality during follow up. However, more recent studies showed that ventricular tachycardia post infarction, regardless of timing, was associated with a higher mortality. Certainly a sustained ventricular tachycardia occurring later than 48 hours post infarction in a patient who is otherwise stable is predictive of a higher risk for sudden cardiac arrest during follow-up. ■

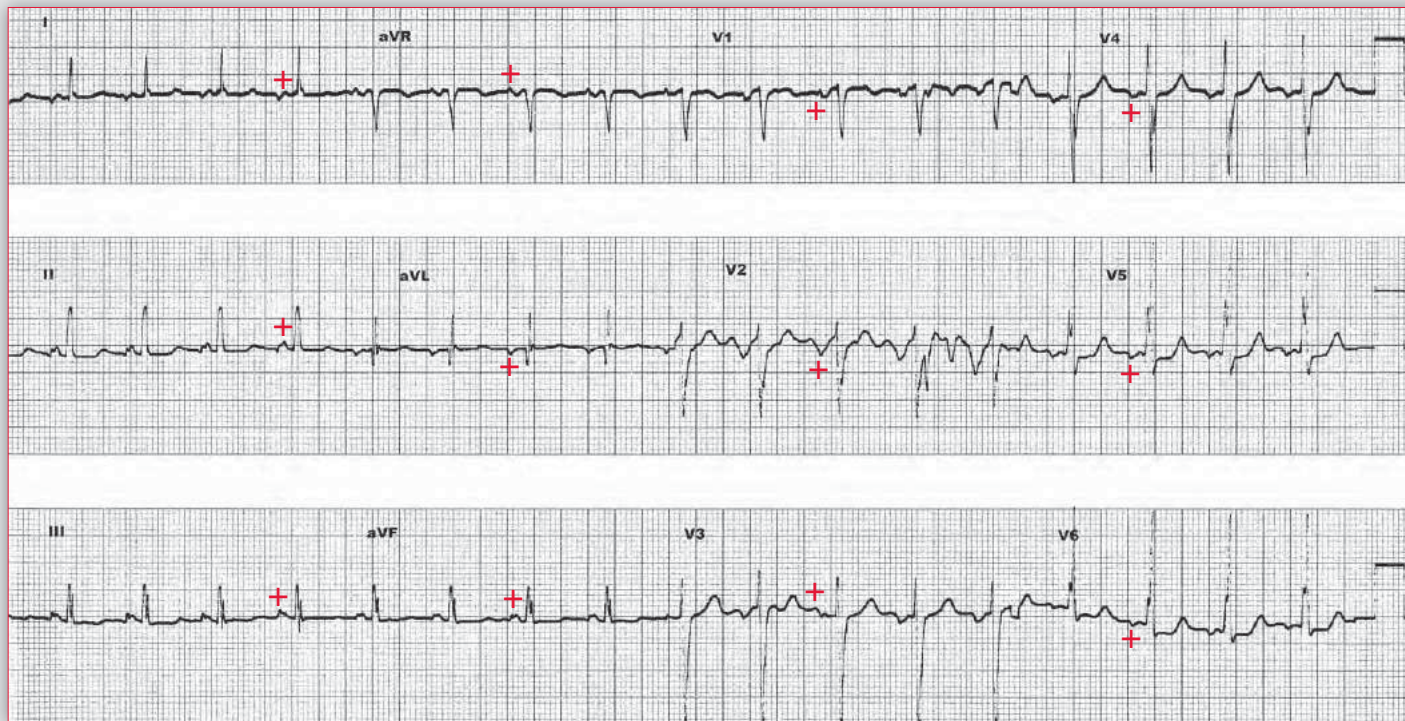
Notes

Practice Case 111

A 60-year-old patient with chronic obstructive pulmonary disease presents with an acute exacerbation. The following ECG is obtained.



Podrid's Real-World ECGs



ECG 111 Analysis: Ectopic atrial tachycardia, nonspecific ST abnormalities

ECG 111 shows a regular rhythm at a rate of 104 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). Nonspecific ST abnormalities are seen in leads V5 and V6. The QT/QTc intervals are normal (340/450 msec). There is a P wave (+) before each QRS complex with a stable PR interval (0.14 sec). The P waves have an abnormal morphology, *ie*, they are biphasic (negative-positive) in leads I, II, and aVF while they are negative in leads V2–V6. Hence this is not a sinus tachycardia, but an ectopic atrial tachycardia.

The normal sinus P wave should be positive in leads I, II, aVF, and V4–V6. P waves that are inverted in any of these leads, or a biphasic, *ie*, negative-positive, are not the result of a sinus rhythm, but rather are

originating from an ectopic focus within the atria. An atrial rhythm (rate < 100 bpm) or an atrial tachycardia (rate > 100 bpm) may represent an escape rhythm, occurring when the sinus rate is slow, or an enhanced ectopic rhythm. In general, such arrhythmias are not serious and do not require therapy, although if there is an atrial tachycardia with a rapid ventricular rate associated with symptoms, rate slowing may be necessary. If there is a suggestion that the rapid rate is a result of sympathetic nervous system activation or elevated circulating catecholamines, a β -blocker may work to slow the rate of ectopic focus discharge. However, most often rate control is achieved with AV nodal blocking agents. Suppression of the atrial ectopic focus will generally require therapy with a class IA, IC, or III antiarrhythmic agent that affects atrial myocardium. ■

Notes

Practice Case 112

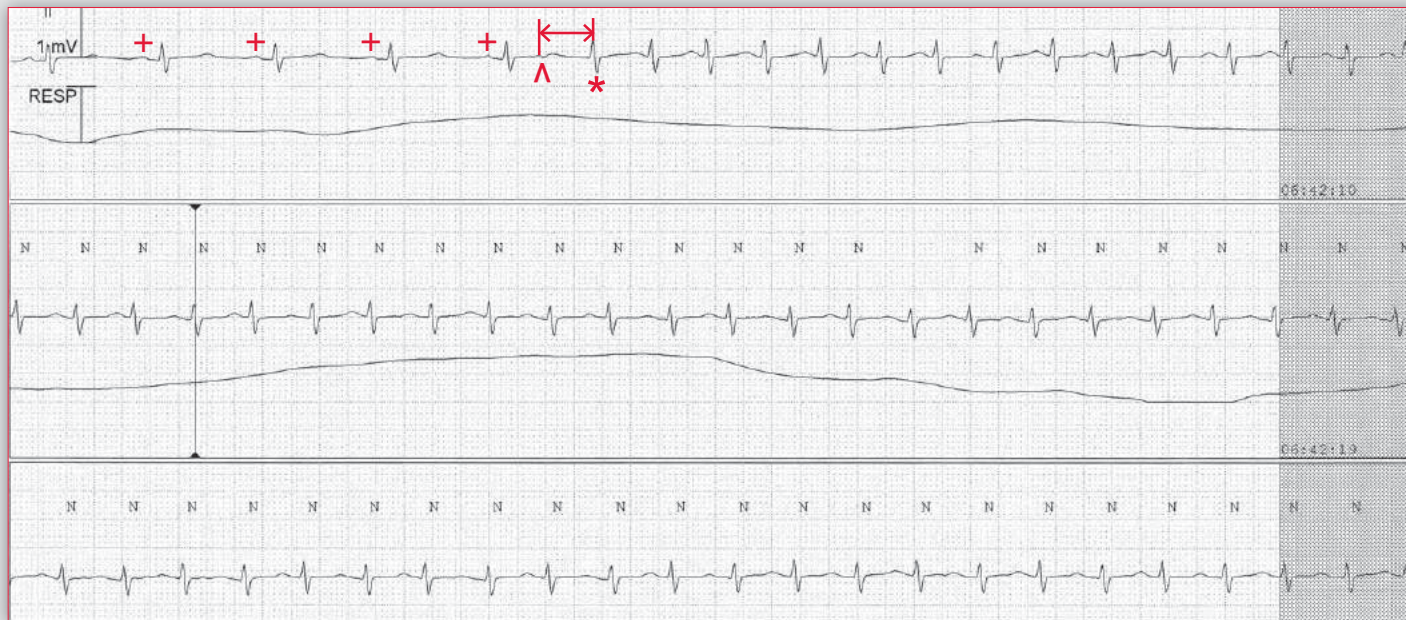
A 47-year-old post-menopausal woman presents with new-onset palpitations. She is placed on telemetry and a rhythm strip showing the onset of a tachycardia is obtained.

What in the ECG precipitates the arrhythmia?

What is the most likely mechanism for the tachyarrhythmia?



Podrid's Real-World ECGs



ECG 112 Analysis: Sinus rhythm, premature atrial complex, narrow complex supraventricular tachycardia, no-RP tachycardia, atrioventricular nodal reentrant tachycardia

ECG 112 rhythm strip shows an initial regular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The fifth QRS complex (*) is premature and is preceded by a P wave (^) and a long PR interval (\leftrightarrow) of 0.36 second. This is a premature atrial complex. Following this premature complex, there is a regular tachycardia at a rate of 140 bpm. There are no P waves before or after the QRS complexes. Hence this is a no-RP tachycardia, and the most likely etiology is a common or typical atrioventricular nodal reentrant tachycardia. This is further supported by the onset of this tachyarrhythmia, *ie*, a premature atrial complex with a long PR interval.

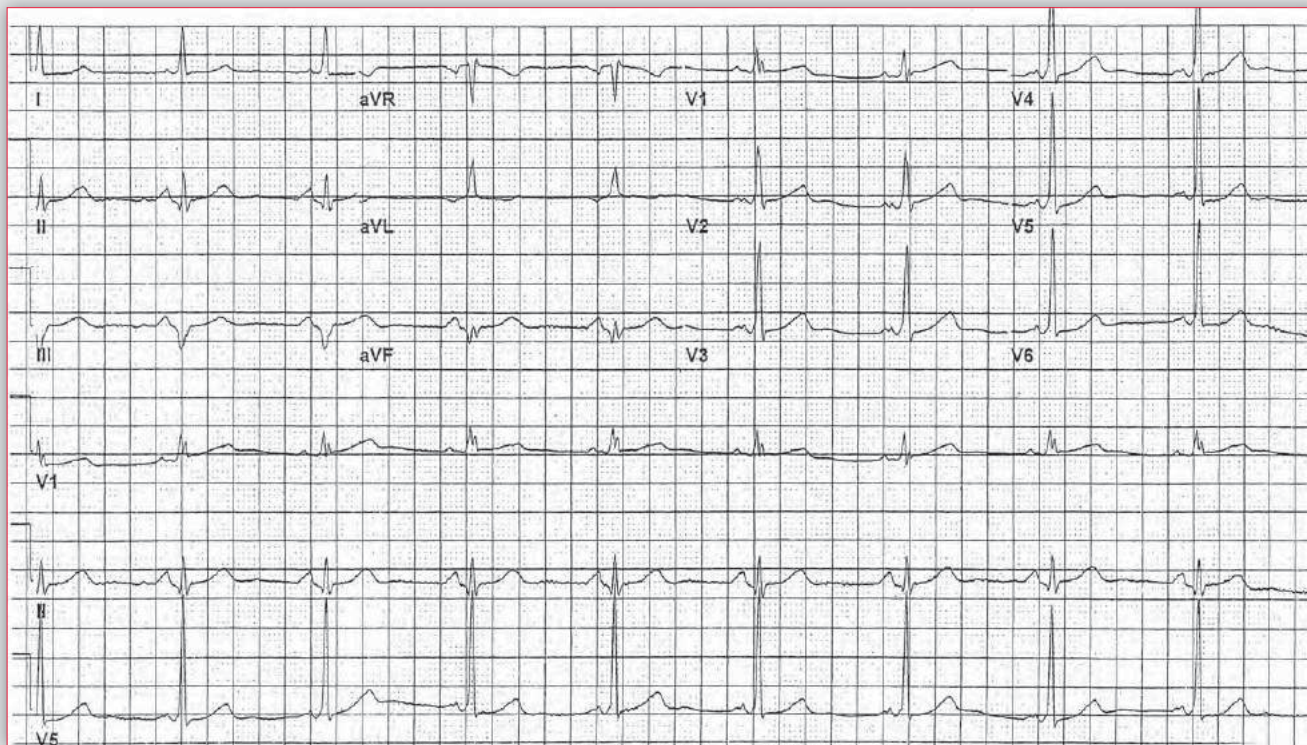
The etiology of an atrioventricular nodal reentrant tachycardia is dual AV nodal pathways. One pathway conducts rapidly but recovers slowly (has a long refractory period). The second pathway conducts slowly, but recovers more quickly (has a short refractory period). These two pathways are proximally and distally within the AV node. During sinus rhythm, the fast pathway predominates and conducts the impulse to the ventricles. If there is a premature atrial complex that reaches the AV node before the fast pathway has recovered, conduction is shifted

to the slow pathway (which recovers more quickly). As a result of the slow conduction through this pathway, the PR interval is prolonged. If the impulse via the slow pathway reaches the distal portion of the circuit at a time when the fast pathway has recovered, the impulse enters this pathway and is rapidly conducted retrogradely to the atria, simultaneous with the antegrade conduction via the His-Purkinje system to the ventricles. Therefore there is simultaneous activation of the atria and ventricles, resulting in a no-RP tachycardia. If the impulse reaches the proximal portion of the circuit at a time when the slow pathway has recovered, the impulse will reenter this pathway. Continuation of this process will result in an atrioventricular nodal reentrant tachycardia. ■

Practice Case 113

An otherwise healthy 48-year-old individual undergoes an electrophysiology procedure for intermittent palpitations and prior syncope. An ECG is obtained prior to the procedure (ECG 113A).

ECG 113A

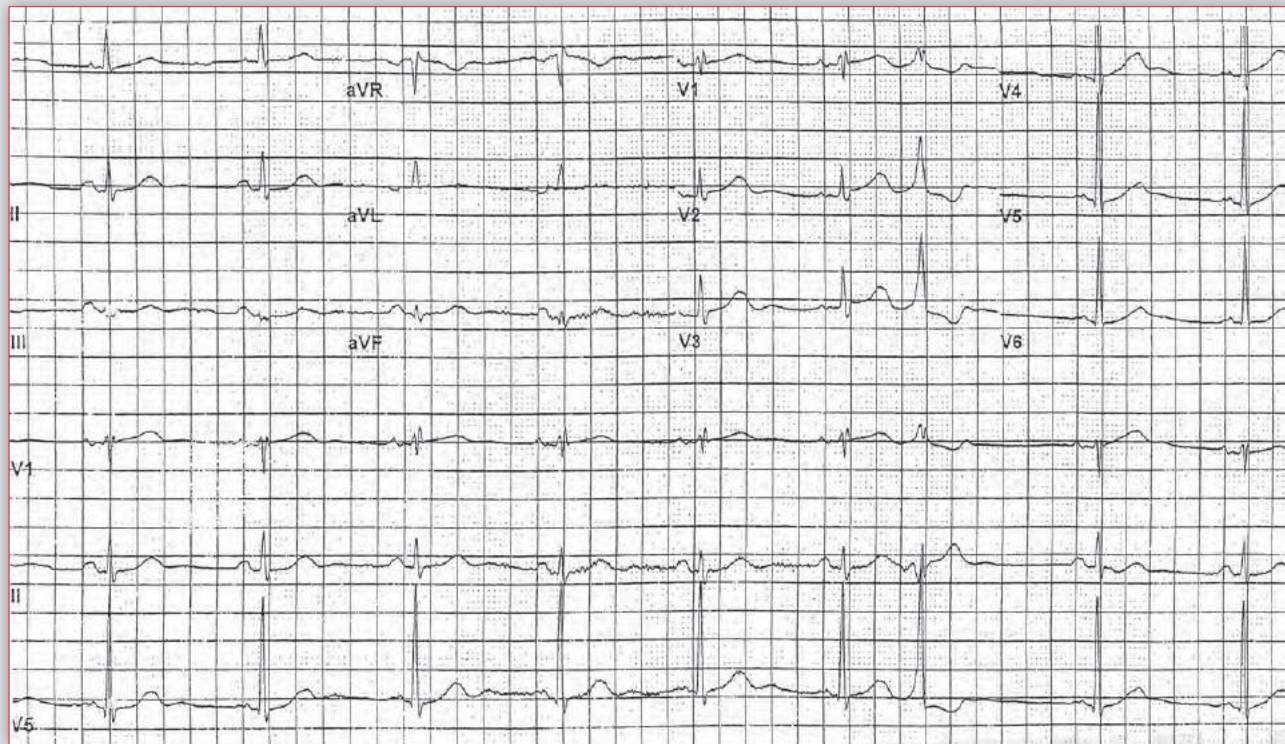


Practice Case 113

Despite the procedure, the patient continues to have symptoms of palpitations. A follow-up ECG is obtained (ECG 113B).

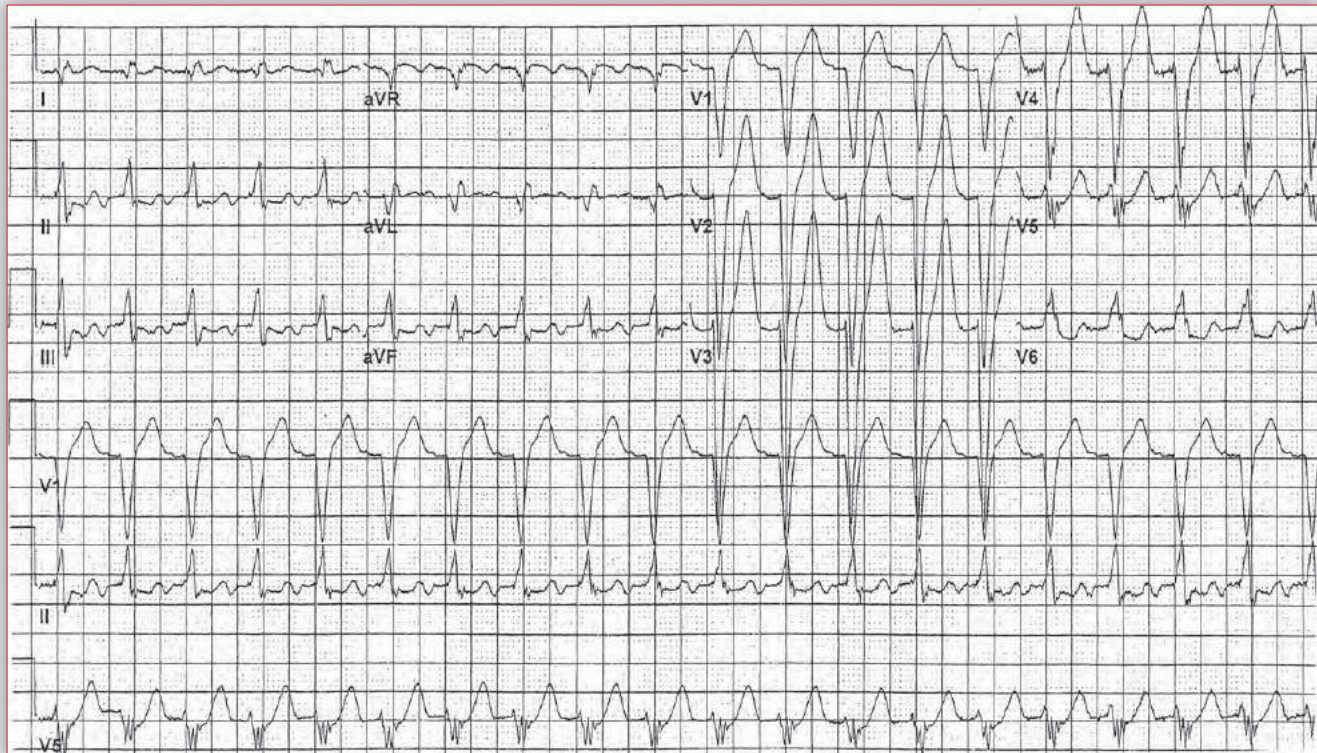
The patient presents to an emergency department during an episode of palpitations and an ECG is obtained (ECG 113C).

ECG 113B



Practice Case 113

ECG 113C



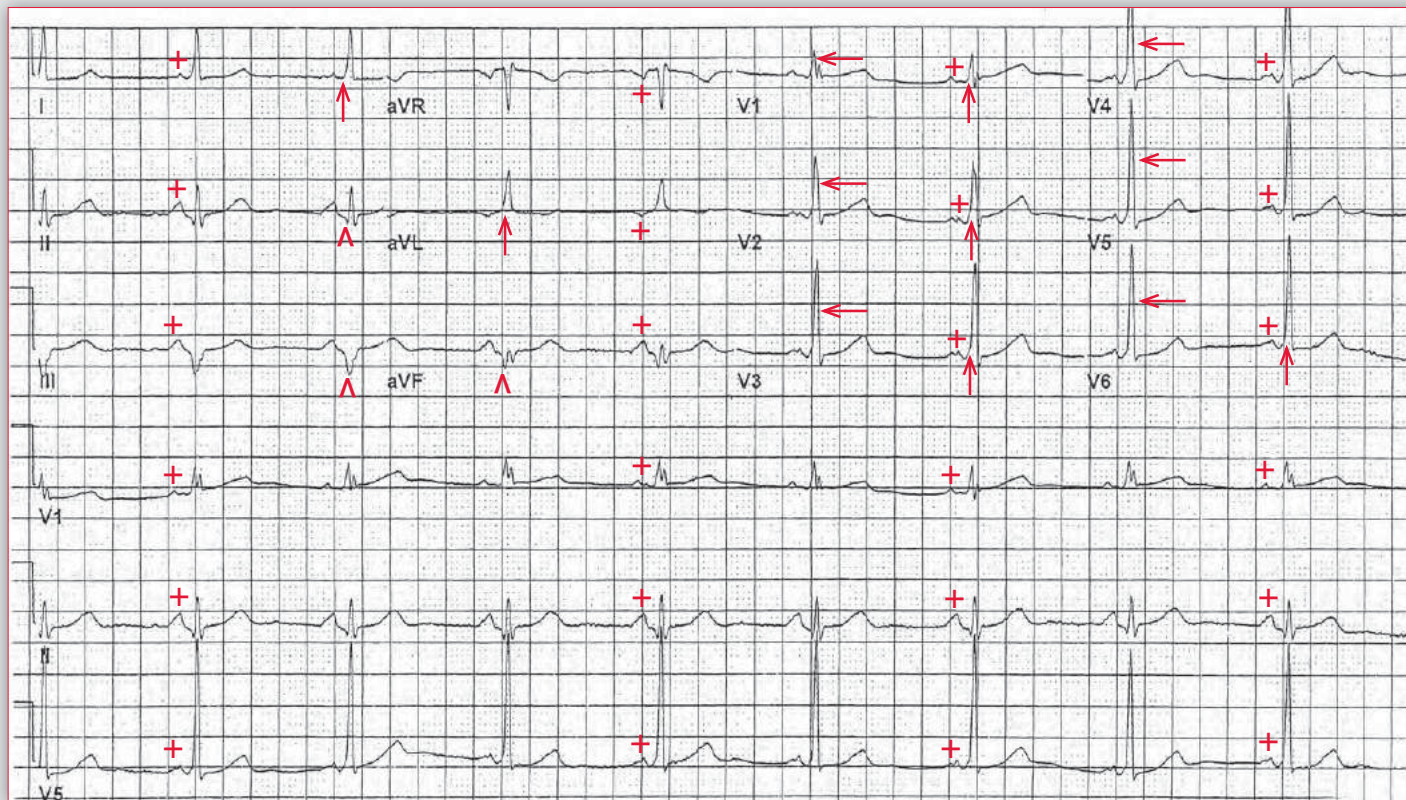
Practice Case 113

What abnormality is seen in ECG 113A?

What does ECG 113B show?

What is the etiology for the palpitations?

Podrid's Real-World ECGs



ECG 113A Analysis: Sinus bradycardia, Wolff-Parkinson-White pattern, posteroseptal accessory pathway, left atrial hypertrophy

ECG 113A shows a regular rhythm at a rate of 56 bpm. There are P waves before each QRS complex (+) with a stable PR interval (0.14 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus bradycardia. However, the P waves are broad, with a prominent notching in leads V2–V5, consistent with left atrial hypertrophy (P-mitrale) or a left atrial abnormality.

The QRS complex duration is increased (0.12 sec), and there is positive concordance across the precordium (tall R wave V1–V6) (←). In addition, the upstroke of the QRS complex is slurred (↑), and this is a delta wave, consistent with a diagnosis of Wolff-Parkinson-White (WPW) pattern. The delta wave indicates direct ventricular myocardial activation via the accessory pathway. As a result of direct myocardial activation, bypassing the normal His-Purkinje system, impulse conduction is slow, accounting for the slurred upstroke. In addition, since initial ventricular activation is via the accessory pathway and not via the normal AV node, the PR interval in WPW is usually very short. In this case, the PR interval is normal, but this is due to a broad P wave; the PR segment is very short, and indeed nonexistent. The PR interval

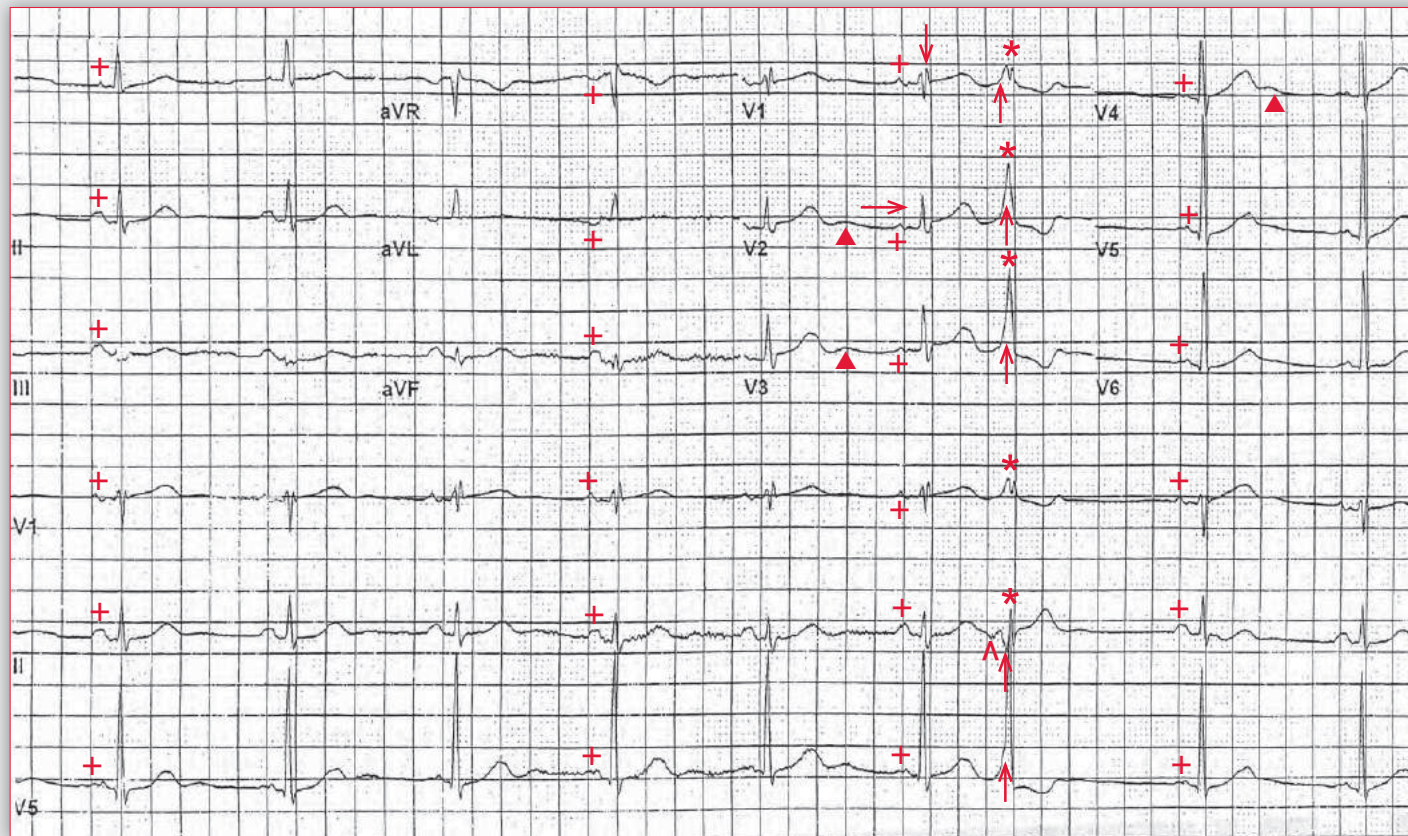
is a result of the P wave (time for atrial depolarization) as well as the PR segment (representing impulse conduction through the AV node–His-Purkinje system). In WPW, the reason for the short PR interval is the very short PR segment.

There are Q waves in leads II, III, and aVF (^), consistent with a pseudo-inferior infarction pattern. As initial ventricular activation is via the accessory pathway, resulting in direct myocardial activation, abnormalities of the left ventricle, including a chronic myocardial infarction, cannot be reliably interpreted. Hence this is termed a pseudo infarction.

The presence of a pseudo inferior wall myocardial infarction pattern means that the accessory pathway is posteroseptal. The presence of a positive delta wave in lead V1 localizes the pathway to the left side with the impulse directed anteriorly towards lead V1 (termed type A). The QT/QTc intervals are prolonged (480/465 msec) but are normal when the prolonged QRS complex duration is considered (460/435 msec).

continues

Podrid's Real-World ECGs



ECG 113B Analysis: Sinus bradycardia, premature atrial complex with preexcitation, left atrial hypertrophy, counterclockwise rotation, U waves

ECG 113B is from the same patient as ECG 113A and was obtained after ablation of the accessory pathway. There is a regular rhythm at a rate of 56 bpm, although the seventh QRS complex is premature (*). There is a P wave before this premature complex (^), and it is negative in the lead II rhythm strip. Hence this is a premature atrial complex. There are P waves before each of the other QRS complexes (+) and the P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus bradycardia. The PR interval is normal (0.18 sec). The QRS complex duration is normal (0.10 sec), and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). There is an R' in lead V1 (↓), consistent with a right ventricular conduction delay. There is also a tall R wave in lead V2 (→) due to early transition or counterclockwise rotation. This represents the axis in the horizontal plane and is established by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces occur early in the precordial leads. The QT/QTc intervals are normal (440/425 msec). U waves are seen after the T waves in leads V2–V4 (▲).

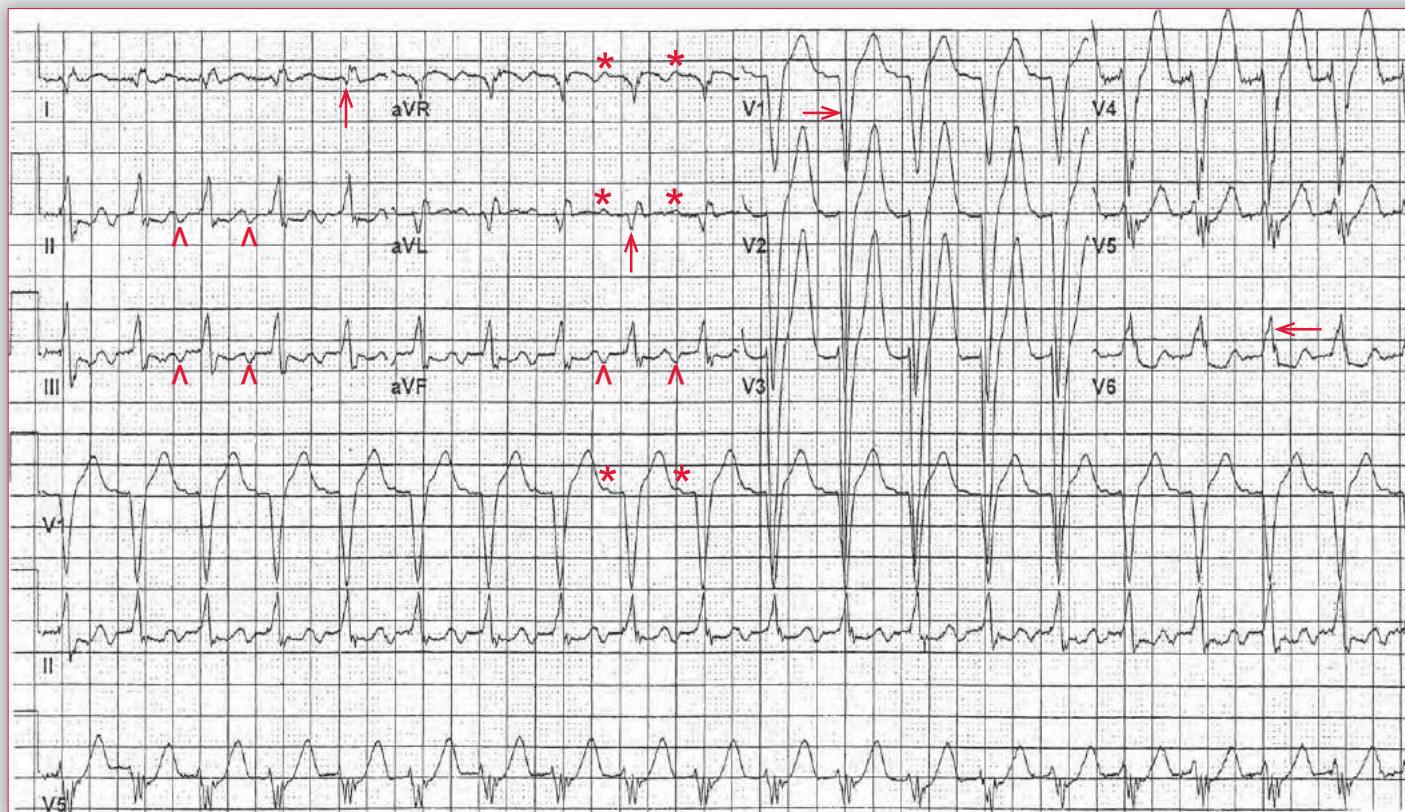
The premature atrial complex is wide (0.14 sec) and has a tall R wave in V1–V3. This is not typical for a right bundle branch block, but is more likely the result of direct myocardial activation. This is not a

ventricular complex as there is a P wave before this QRS complex (^), *ie*, it is a premature atrial complex. The upstroke of this complex is slower (slurred), consistent with a delta wave (†). Indeed, the QRS complex is identical in morphology to the preexcited complexes seen in ECG 113A, although it is wider and hence more preexcited, *ie*, more of ventricular activation results from impulse conduction through the accessory pathway, while there is less that is due to conduction via the normal AV node–His–Purkinje system. This is due to slowed AV nodal conduction resulting from the premature complex (*ie*, decremental conduction in which AV nodal conduction is slower when it is being stimulated at a faster rate) or a premature impulse originating close to the accessory pathway. In lead II there is a Q wave, and this is a pseudo inferior wall infarction pattern, similar to what was seen in ECG 113A. Importantly, the normal, non-preexcited QRS complexes do not show an inferior wall myocardial infarction.

The presence of preexcitation with the premature complex means that the accessory pathway is still active, although the preexcitation is not seen with normal sinus rhythm.

continues

Podrid's Real-World ECGs



ECG 113C Analysis: Long RP tachycardia, orthodromic atrioventricular reentrant tachycardia, intraventricular conduction delay

ECG 113C shows a regular rhythm at a rate of 120 bpm. The QRS complex duration is increased (0.14 sec) and the morphology resembles a left bundle branch block with a QS complex in lead V1 (\rightarrow) and a broad R wave in lead V6 (\leftarrow). However, there is a Q wave in leads I and aVL (\uparrow), indicating initial septal depolarization. Septal Q waves are not seen with a left bundle branch block, as septal activation originates from a median or septal branch which comes from the left bundle. Therefore this is an intraventricular conduction delay. The QT/QTc intervals are prolonged (360/510 msec) but are normal when the prolonged QRS complex duration is considered (320/445 msec).

Although there are no obvious P waves seen before or after any of the QRS complexes, there are negative waveforms seen in leads II, III, and aVF (\wedge); these waveforms are positive in leads aVR, aVL, and V1 (\ast). Hence these are P waves (RP = 0.30 sec and PR = 0.20 sec), and therefore this is a long RP tachycardia. Etiologies for a long RP tachycardia include sinus tachycardia (eliminated as a diagnosis as the P waves are negative in leads II and aVF), atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, an atypical atrioventricular nodal reentrant tachycardia (termed fast-slow, *ie*, antegrade conduction to the ventricle via the fast pathway and retrograde atrial activation via the slow pathway), and an atrioventricular reentrant tachycardia (AVRT). Given the known diagnosis of WPW, the most likely etiology is atrioventricular reentrant tachycardia (AVRT). Most often, an AVRT is associated with a short RP interval. However, as this patient has had an accessory pathway ablation, it is likely that the resulting changes in accessory pathway conduction characteristics cause the RP interval to be long.

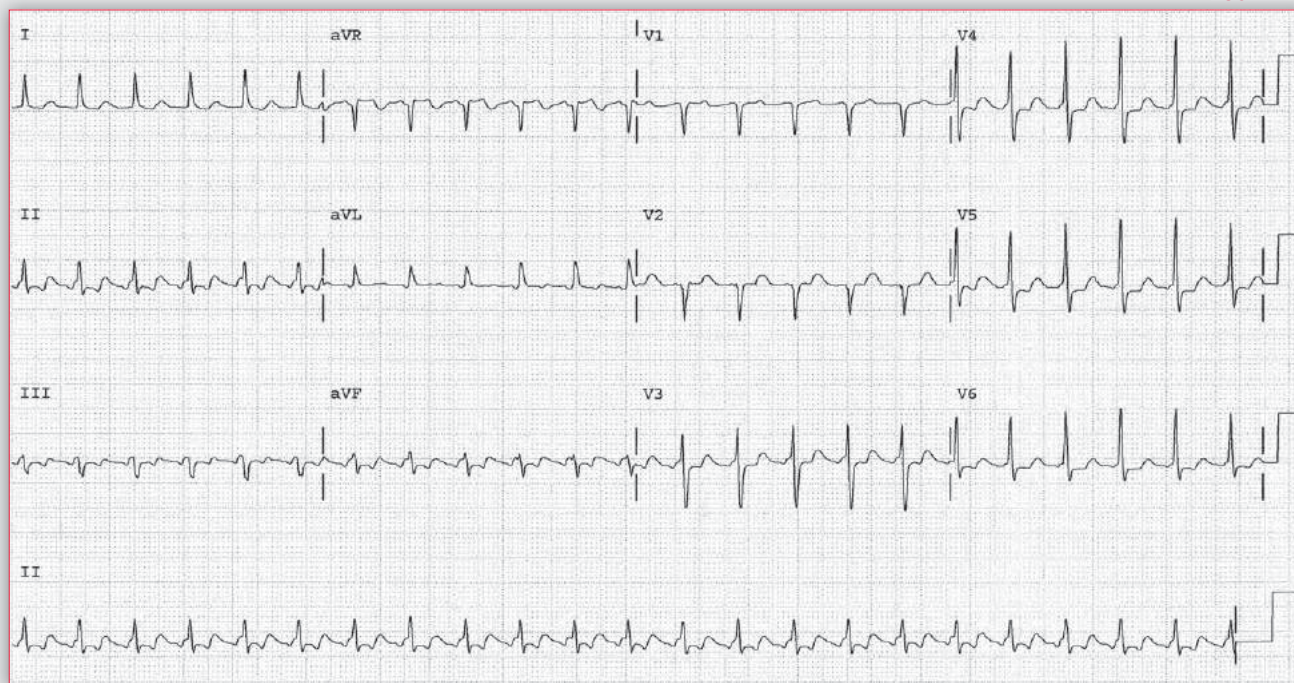
An AVRT may have two different manifestations. Most often, the QRS complexes are narrow and normal in morphology, and this is termed an orthodromic AVRT, resulting from ventricular activation or AV conduction via the AV node–His-Purkinje system and retrograde atrial activation (VA conduction) via the accessory pathway. Less commonly seen is an antidromic AVRT, in which the QRS complexes are wide. This is due to the fact that the antegrade activation of the ventricles (AV conduction) is via the accessory pathway, while retrograde activation of the atria (VA conduction) is via the AV node–His-Purkinje system. In this situation, the wide QRS complexes are due to preexcitation from activation via the accessory pathway, and they will resemble the preexcited complexes during sinus rhythm, although they are often wider, or more preexcited, since all of ventricular activation is via the accessory pathway (in contrast to the sinus complexes, which are usually fusion representing ventricular activation via the accessory pathway and the normal AV node–His-Purkinje pathway).

In this case, the wide QRS complexes do not have the same morphology as the preexcited QRS complexes from the baseline ECG 113A or the preexcited premature atrial complex seen in ECG 113B. Hence this is an orthodromic AVRT. The wide QRS complexes are the result of a rate-related intraventricular conduction delay, as the QRS complexes in ECG 113B are narrow with a normal morphology. ■

Practice Case 114

A 42-year-old woman with exophthalmos, recent weight loss, and hand tremor presents with new-onset palpitations. An ECG (ECG 114A) is obtained and compared to a previous ECG from a year before (ECG 114B). She is subsequently diagnosed with Graves' disease.

ECG 114A

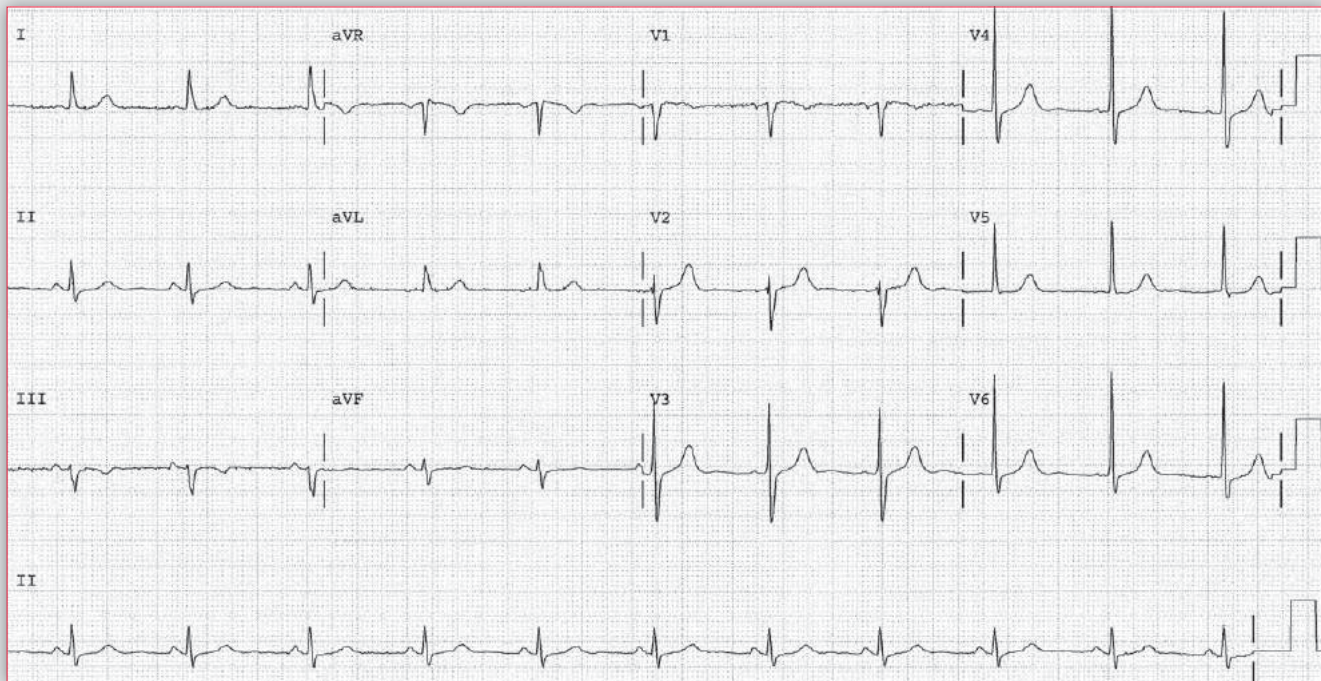


Practice Case 114

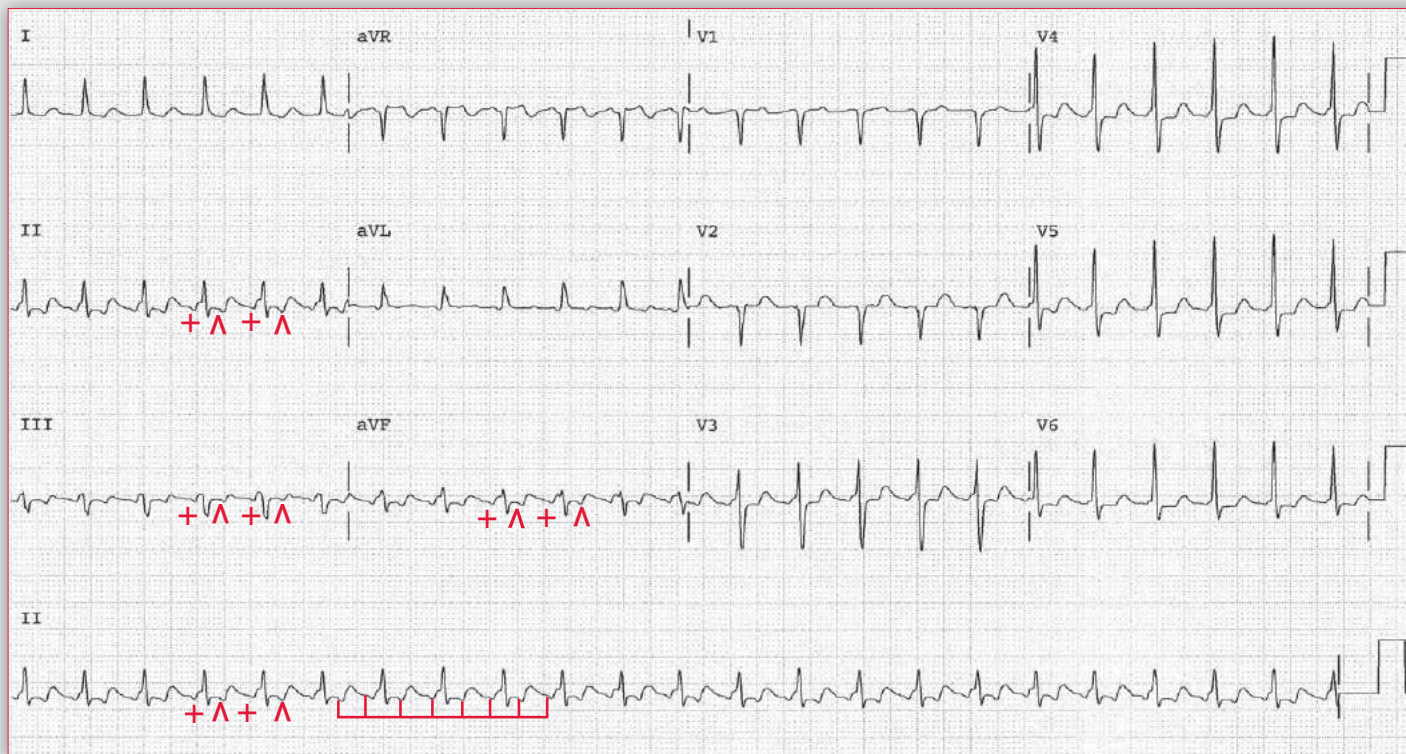
What is the rhythm abnormality?

What other cardiac conditions are associated with Graves' disease?

ECG 114B



Podrid's Real-World ECGs



ECG 114A Analysis: Narrow complex supraventricular tachycardia, long RP tachycardia, atrial flutter with 2:1 AV block

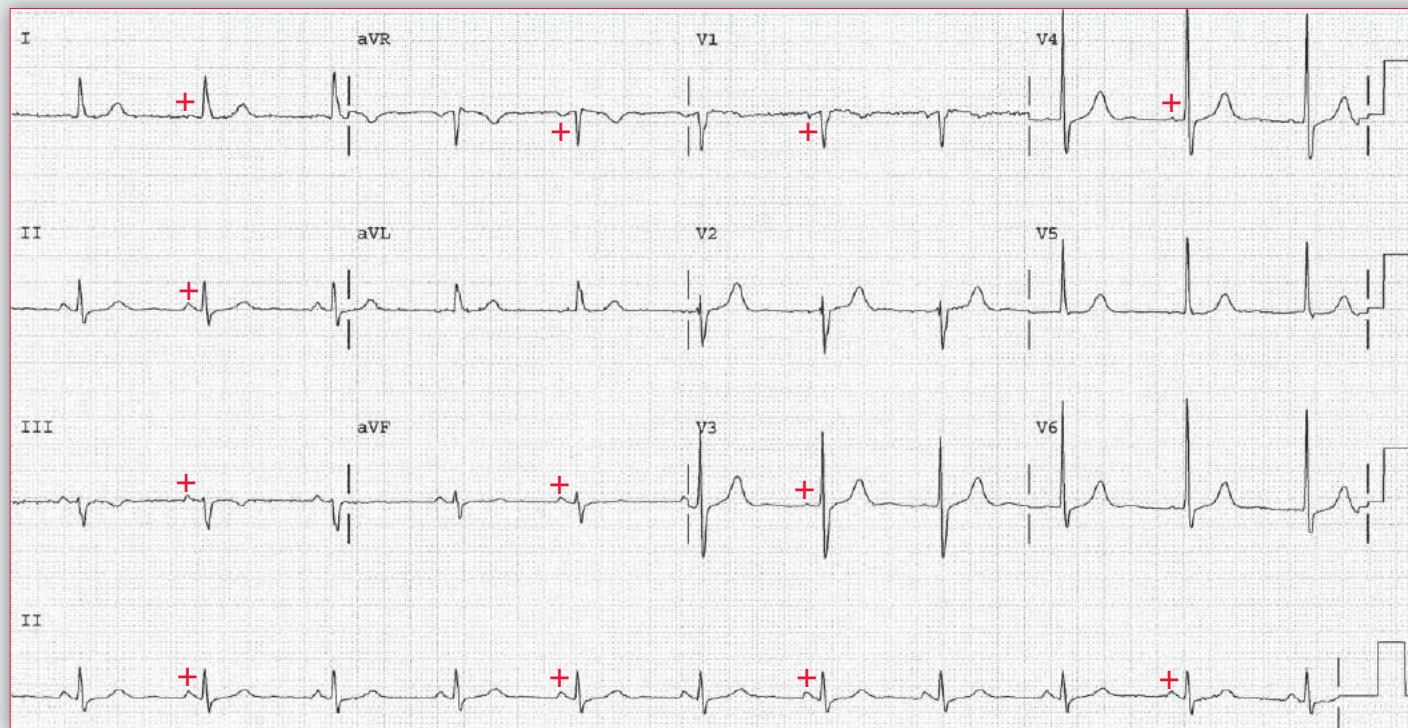
ECG 114A shows a regular rhythm at a rate of 138 bpm. The QRS complex duration (0.08 sec) and morphology are normal with an axis of about 0° (positive QRS complex in lead I and biphasic in lead aVF). The QT/QTc intervals are normal (300/450 msec). There are no distinct P waves before or after any QRS complex, but there is evidence of atrial activity seen before each QRS complex, especially in leads II, III, aVR, and aVF (+). The waveforms are negative. This appears to be a long RP tachycardia. However, with close inspection of these leads, a second waveform of the same morphology can be seen at the very end of the QRS complex (^). Although it appears that this is ST-segment depression, this waveform has the same morphology as the waveform before the QRS complex, and the intervals between them are identical (\sqcup), at a rate of 280 bpm. The waveforms appear to be undulating (sawtoothed in pattern) without any isoelectric baseline between each waveform. Given the rate of 280 bpm and the morphology of the atrial waveforms, this is atrial flutter with 2:1 AV conduction.

The atrial flutter in Graves' thyrotoxicosis is generally due to the enhanced sympathetic state that is associated with the hyperthyroidism. Treatment with a β -blocker may result in further AV block and rate slowing and might also result in reversion of the arrhythmia. Spontaneous reversion is also likely with treatment of the hyperthyroidism.

In addition to atrial flutter, Graves' thyrotoxicosis can be associated with the following cardiac conditions: inappropriate sinus tachycardia, atrial fibrillation, supraventricular tachycardia, high output heart failure, systolic heart failure, and dilated cardiomyopathy.

continues

Podrid's Real-World ECGs



ECG 114B Analysis: Normal sinus rhythm

ECG 114B is from the same patient as ECG 114A and is the baseline ECG. There is a regular rhythm at a rate of 66 bpm. The QRS complex duration, axis, and morphology are identical to that seen in ECG 114A. The QT/QTc intervals are normal (400/420 msec). There are P waves seen before each QRS complex (+) with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. By comparison with ECG 114A, it can be seen that the ST segments are now normal, consistent with the fact that the ST-segment abnormalities in ECG 114A were not the

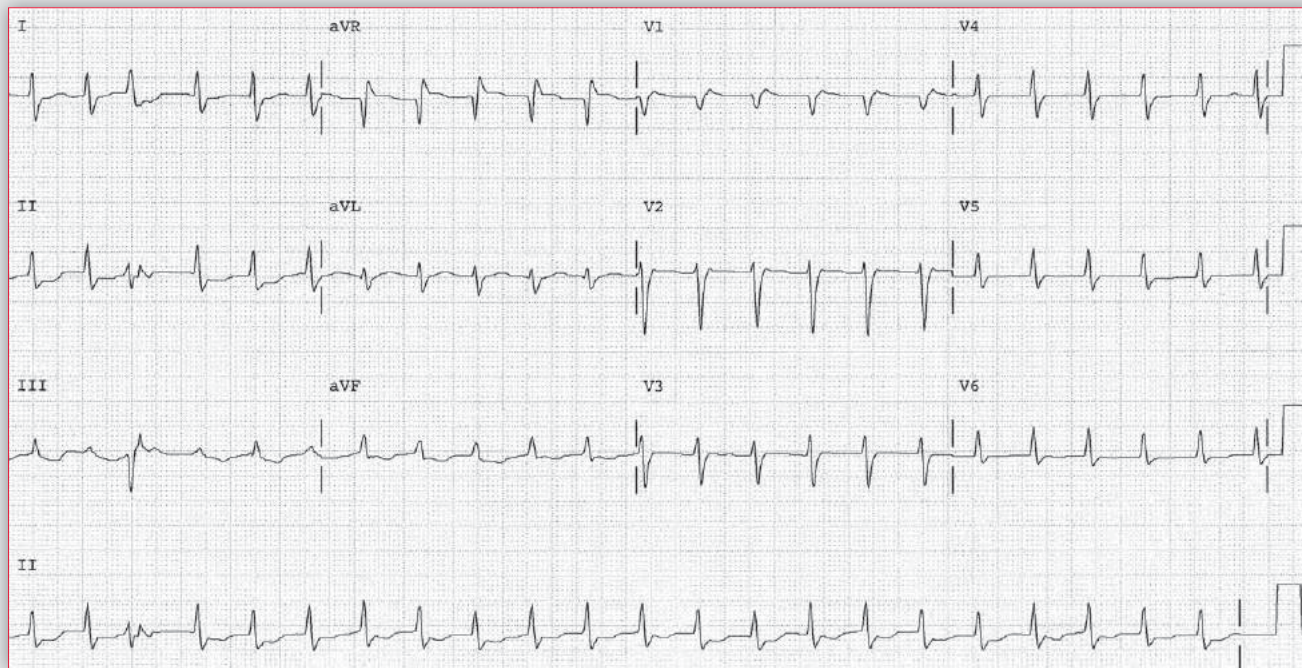
normal morphology of the QRS complex and were indeed embedded flutter waves.

Atrial flutter is often a difficult diagnosis to establish and is often overlooked because one of the two flutter waves may be superimposed on the end of the QRS complex, resembling an S wave or ST-segment depression or at the beginning of the QRS complex, resembling a Q wave. ■

Practice Case 115

A 62-year-old man with a history of congestive heart failure and symptomatic paroxysmal atrial fibrillation treated chronically with atenolol and digoxin presents with palpitations and dizziness to the emergency department. In the review of systems, the patient describes dark-colored,

ECG 115A

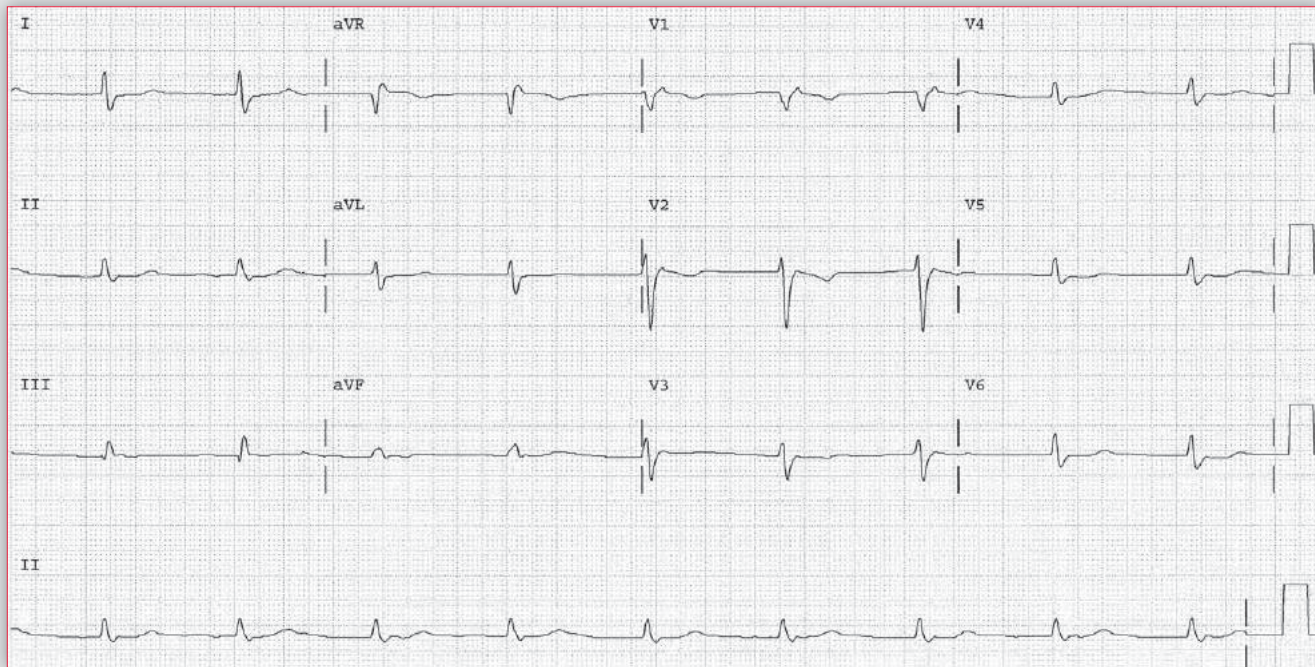


Practice Case 115

concentrated urine over the past few days. The patient also reports low back pain in recent times, for which he takes ibuprofen three times daily. An ECG is obtained (ECG 115A).

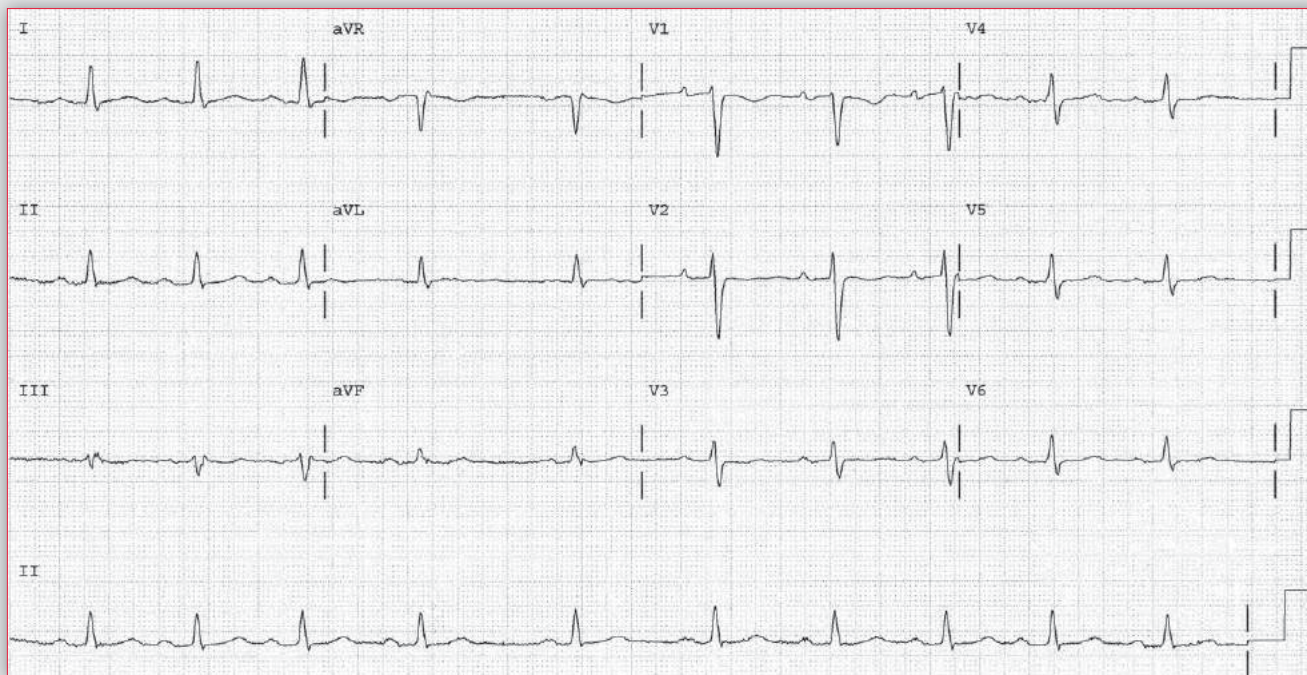
ECG 115B is obtained during the patient's recovery and ECG 115C is the patient's baseline ECG.

ECG 115B



Practice Case 115

ECG 115C



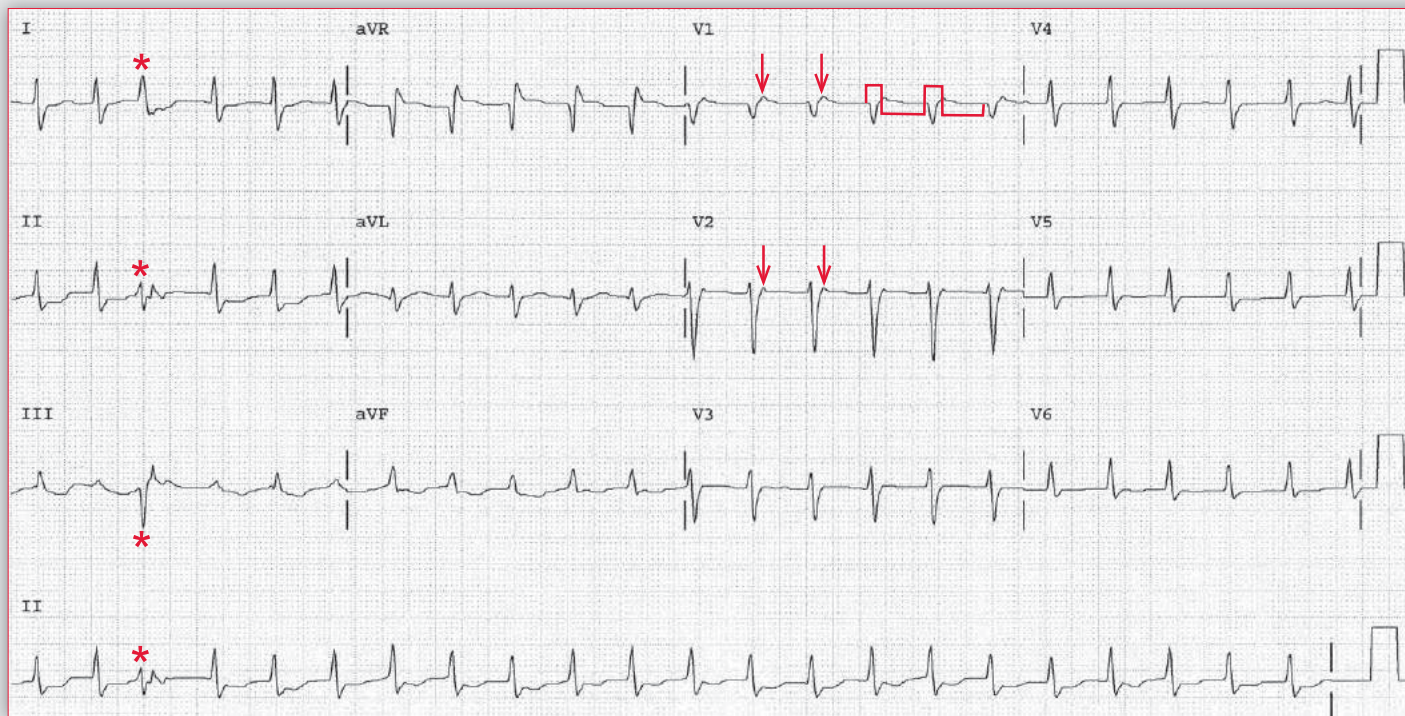
Practice Case 115

What is the rhythm abnormality for the first tracing (ECG 115A)?

**Based on the other two tracings (ECG 115B and ECG 115C),
what is the rhythm abnormality noted?**

What is the most likely clinical diagnosis?

Podrid's Real-World ECGs



ECG 115A Analysis: Supraventricular tachycardia, short RP tachycardia, junctional ectopic tachycardia, premature ventricular complex

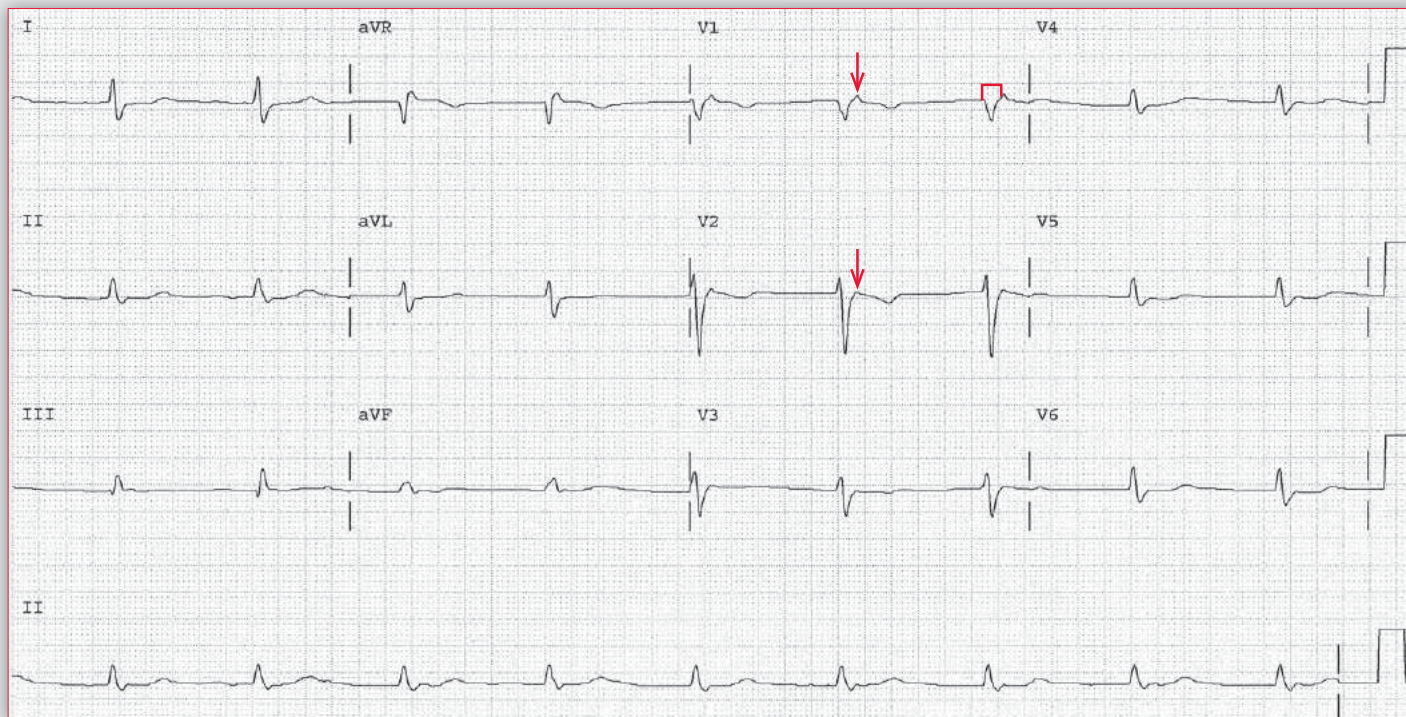
ECG 115A shows a regular rhythm at a rate of 140 bpm. The QRS complex duration is normal (0.10 sec), and there is a normal axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/430 bpm). There is one premature QRS complex (*) that has a wider duration and an abnormal morphology; this is a premature ventricular complex.

There are no P waves seen before or after any QRS complex. However, there is what appears to be an R' in leads V1–V2 (↓). It is broader than a usual R' waveform and it does not appear to be superimposed at the terminal portion of the QRS complex, but rather it occurs after the terminal portion of the QRS complex, within the initial part of the ST segment. This is therefore a P wave and hence this is a short

RP tachycardia with an RP interval of 0.14 sec (□) and PR interval of 0.32 second (⊐). Etiologies include sinus tachycardia, atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, atrioventricular reentry tachycardia, or an uncommon form of a common or typical atrioventricular nodal reentrant tachycardia (AVNRT), termed slow-slow. The common form of an AVNRT is termed slow-fast, *ie*, the antegrade conduction to the ventricle is via the slow pathway, while the retrograde conduction back to the atria is via the fast pathway, resulting in a no-RP tachycardia. If there is slowing of conduction in the fast pathway, as a result of age-related changes in the tract or the effect of a drug, retrograde conduction may be slowed, resulting in a short RP tachycardia, and hence termed slow-slow.

continues

Podrid's Real-World ECGs



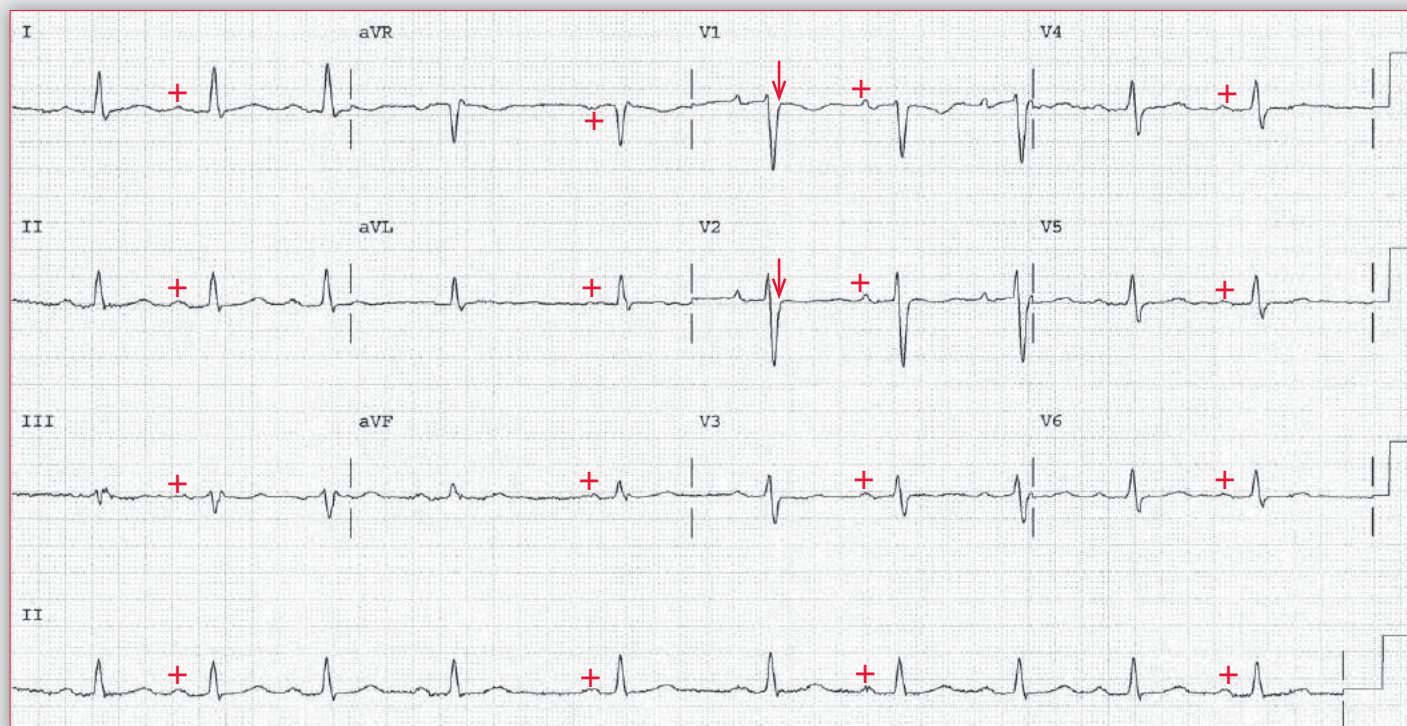
ECG 115B Analysis: Junctional ectopic rhythm

ECG 115B is from the same patient as ECG 115A. There is a regular rhythm at a rate of 56 bpm. The QRS complex duration, morphology, and axis is the same as in ECG 115A. The QT/QTc intervals are the same (460/440 msec). There are no P waves seen before or after any QRS complex. The QRS morphology in V1–V2 is identical to that seen in ECG 115A and there is the same positive waveform seen immediately after the QRS complex (↓). Although resembling an R', it is broad and is not simultaneous with the end of the QRS complex, but after it within

the ST segment; hence this is a P wave. The RP interval (□) is the same as seen in ECG 115A. Therefore this is a junctional ectopic rhythm, establishing the fact that the rhythm in ECG 115A is a junctional ectopic tachycardia. The rhythm is the same, originating from an ectopic focus within the AV node or junction. When the rate is < 100 bpm, it is called a junctional rhythm, whereas when the rate is > 100 bpm, it is termed a junctional tachycardia.

continues

Podrid's Real-World ECGs



ECG 115C Analysis: Sinus rhythm, sinus arrhythmia, first-degree AV block

ECG 115C is from the same patient as ECG 115A and 115B. It is the baseline ECG for the patient. There is an irregularly irregular rhythm with an average rate of 66 bpm. There are three arrhythmias that are irregularly irregular. These are sinus arrhythmia, in which there is one P-wave morphology and PR interval; a multifocal atrial rhythm (rate < 100 bpm) or a multifocal atrial tachycardia (rate > 100 bpm), in which there are > 3 different P-wave morphologies with no dominant P-wave morphology and variable PR intervals; and atrial fibrillation, in which there is no organized atrial activity or P wave. There is a P wave (+) before each QRS complex with a stable P-wave morphology and PR interval (0.26 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus arrhythmia with first-degree AV block. Noted is the absence of the R' in leads V1 and V2 (↓), supporting the fact that the R' was a P wave and not a true R'. This confirms that the rhythm in ECG 115A was a junctional ectopic tachycardia and the rhythm in ECG 115B was a junctional ectopic rhythm.

The clinical scenario points towards digitalis toxicity from an increase in the digoxin blood level due to renal insufficiency from chronic NSAID use. Digitalis toxicity may be associated with various rhythm abnormalities, including sinus bradycardia, ectopic atrial tachycardia with or without AV block, an accelerated junctional rhythm, a junctional ectopic tachycardia, bidirectional (junctional) tachycardia, ventricular tachycardia (ectopic), or ventricular fibrillation. The etiology of digoxin toxicity arrhythmias is related to a marked increase in vagal tone as well as an increase in central sympathetic neural output.

As vagal tone increases, there is a decrease in sinus node rate. The increasing central autonomic tone results in acceleration of an ectopic atrial focus; if the rate of this focus is faster than the sinus rate, there is a non-paroxysmal ectopic atrial tachycardia. As the atrial rate increases, there may be AV block, related to the enhanced vagal effect on AV nodal conduction. This is termed atrial tachycardia with block. As the atrial rate increases, all conduction through the AV node may be blocked (resulting from enhanced vagal tone), and therefore complete heart block with a junctional escape rhythm may develop. With further increase in digoxin levels and increase in sympathetic outputs, the junctional rate may accelerate, resulting in a nonparoxysmal junctional tachycardia. With a further increase in digoxin level, there is slowing of conduction through the bundles, resulting in a junctional tachycardia with alternating left and right bundle branch block, termed bidirectional tachycardia. If complete block through the bundles occurs, there is an escape ventricular focus and ventricular tachycardia. Importantly, these arrhythmic abnormalities are not the result of reentry, but are the result of enhanced automaticity or triggered activity. Hence cardioversion will not be affected and should not be performed, as the myocardium is very sensitive to electrical stimulation and ventricular fibrillation may be provoked.

Treatment for symptomatic arrhythmias and also bidirectional tachycardia is Digibind. Atrial and junctional tachycardia may respond to β -blockade as the mechanism is related in part to enhanced sympathetic tone. ■

Notes

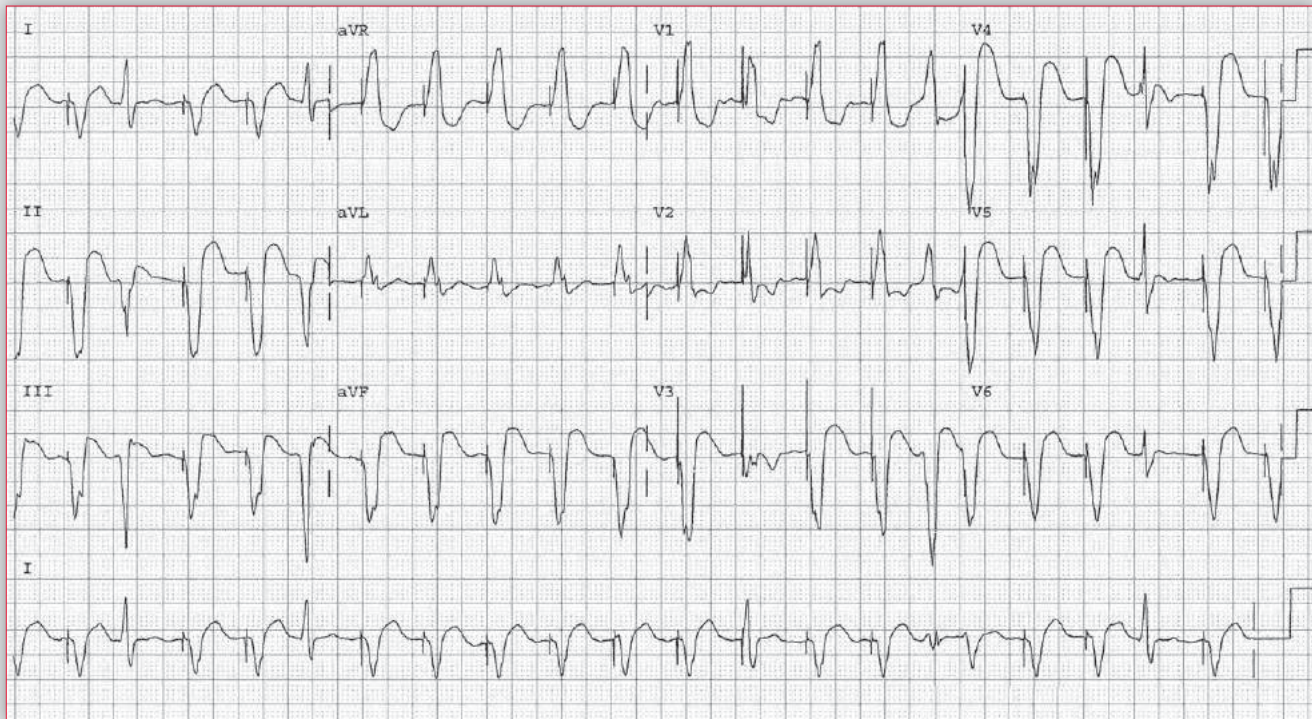
Practice Case 116

A 54-year-old woman with a history of systolic heart failure and dilated cardiomyopathy presents with new-onset dizziness and palpitations. She states that she recently had an implantable pacemaker, but is not aware of any of the details. The following ECG is obtained.

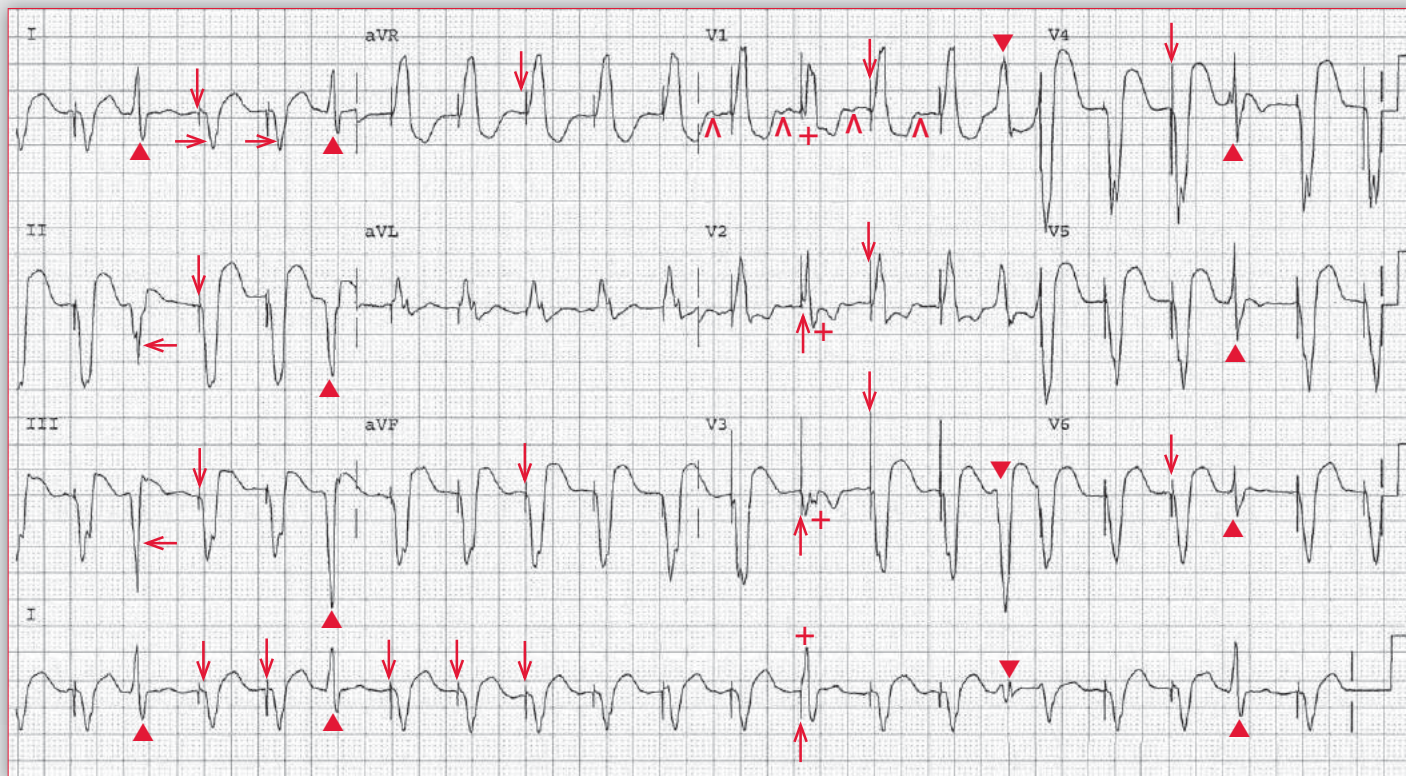
What is the cause of the patient's symptoms?

How can one explain the thirteenth QRS complex?

What type of pacemaker does the patient have?



Podrid's Real-World ECGs



ECG 116 Analysis: Pacemaker-associated tachycardia, atrial fibrillation, biventricular pacing, pseudofusion, premature ventricular complex

ECG 116 shows a regular wide complex tachycardia at a rate of 130 bpm. The QRS complex duration is increased (0.14 sec). There are pacing stimuli seen before most of the QRS complexes (↓). Hence this is a ventricular paced rhythm. Since the ventricular pacing rate is fast at 130 bpm, the pacemaker is dual chamber, sensing the atrium and pacing the ventricle (*ie*, DDD). There are no obvious P waves seen; however, there are irregular undulations of the baseline, best seen in lead V1 (Λ). Hence the underlying rhythm is atrial fibrillation and the pacemaker is thus pacing at its upper rate limit, accounting for the wide complex tachycardia. This is therefore a pacemaker-associated tachycardia.

The third, sixth, thirteenth, and twentieth QRS complexes (+,▲) have a different morphology. They are early, and the third, sixth, and twentieth complexes (▲) are without pacing stimuli. The thirteenth QRS complex (+) has the same morphology but is preceded by a pacemaker stimulus (†). The QRS complex duration is normal (0.10 sec). There are Q waves in leads II and III (←). Hence these are native supraventricular complexes, and there is an old inferior wall myocardial infarction. The underlying rhythm is atrial fibrillation, and if the ventricular response rate increases, the pacemaker output is suppressed, accounting for why these complexes are not paced. The thirteenth QRS complex (+) also has a different QRS morphology, similar to the native QRS complexes, although slightly wider; however, it is not early and there is a pacing stimulus before it (†) that does not capture the ventricle. This is termed

pseudofusion, *ie*, there is a pacing stimulus but it does not result in ventricular activation because the native QRS complex occurs at the same time. The sixteenth QRS complex (▼) is also early, is without a preceding pacemaker stimulus and has a different morphology than the paced complexes and the native QRS complexes. Hence this is a premature ventricular complex.

The standard pacemaker has a lead in the right atrium and right ventricle. Hence the paced ventricular QRS complex has a typical left bundle branch block morphology, as the impulse is originating in the right ventricle with delayed activation of the left ventricle. The morphology consists of a broad R wave in lead I (which is a bipolar lead looking at impulse in a right-to-left direction with a positive deflection if the impulse is directed to the left) and in leads V5–V6 as well as a QS complex in lead V1 (all activation going away from the right). In contrast, the QRS complexes in this ECG have a tall broad R wave in lead V1 and a QS complex in leads V5–V6, which is not typical for a left bundle branch block, but resembles a right bundle branch block. However, a left bundle branch block may also present with QS complexes in leads V5–V6. In addition, a right ventricular lead near the intraventricular septum may be associated with a tall R wave in lead V1. Most important is the QRS complex in lead I that has a QS morphology (→). This is never seen with a left bundle branch block, which has all activation going from right-to-left, and hence there will

continues

be a tall R wave in lead I. A QS morphology in lead I means that the activation is occurring in a left-to-right direction. Therefore, initial activation is occurring in the left ventricle. This is the pattern for left ventricular pacing, consistent with a biventricular pacemaker.

Most pacemakers have a programmable feature known as mode switching. If a rapid atrial rate is sensed, the pacemaker automatically switches to a VVI mode, or a demand pacemaker. If this does not occur and the pacemaker tracks an atrial arrhythmia at its upper rate limit, a magnet can be used in the acute setting to turn off all atrial and ventricular sensing. The pacemaker will no longer track the atrial impulses and will function as a fixed-rate pacemaker. As the native complexes will be seen, the rhythm can be definitively diagnosed and treated as conduction will now be through the normal AV node. However, a

magnet will not affect defibrillator function. Many patients who have biventricular pacemakers typically also obtain automatic implantable cardioverter-defibrillators (AICD) since the two devices have similar clinical indications (*ie*, heart failure with reduced LVEF).

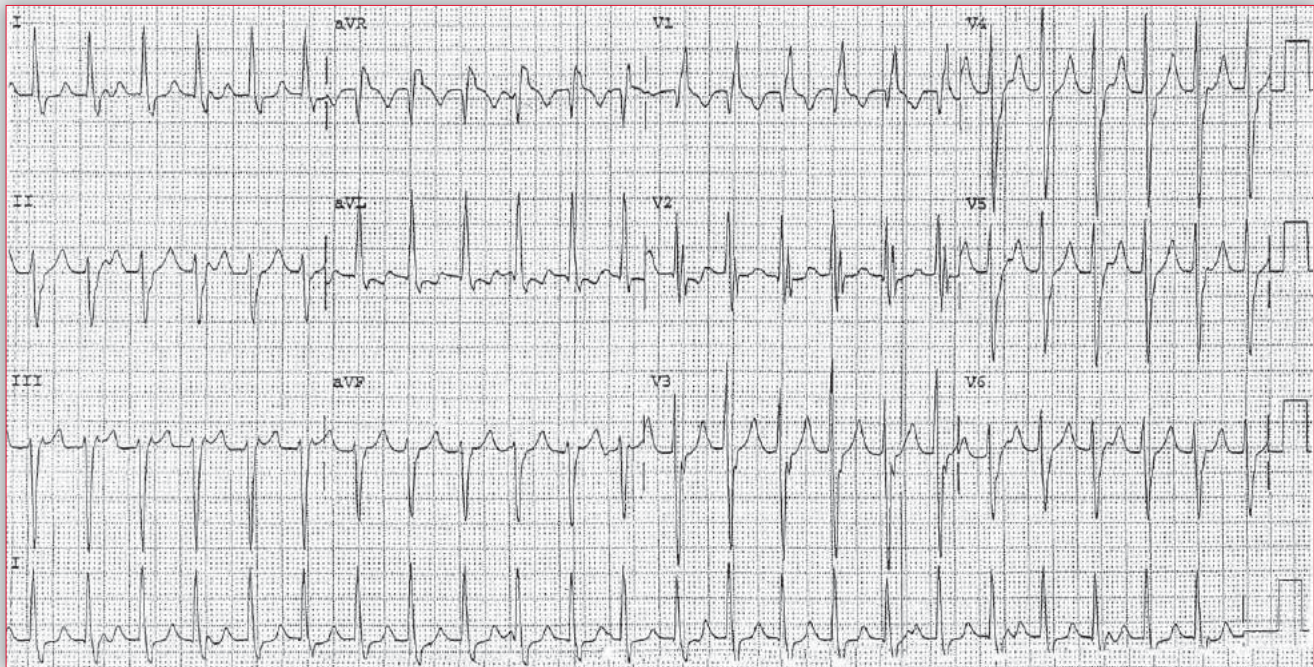
In patients with atrial fibrillation the benefit of biventricular pacing is reduced if there is intact AV nodal conduction and the ventricular rate is faster than the lower rate limit of the pacemaker. Hence the atrial fibrillation should be reverted and prevented or the AV node adequately blocked to prevent the ventricular rate from being excessive and faster than the lower rate limit of the pacemaker. If the patient remains in atrial fibrillation, then atrial sensing should be permanently disabled, with the pacemaker functioning in a VVI mode, *ie*, demand ventricular pacemaker. ■

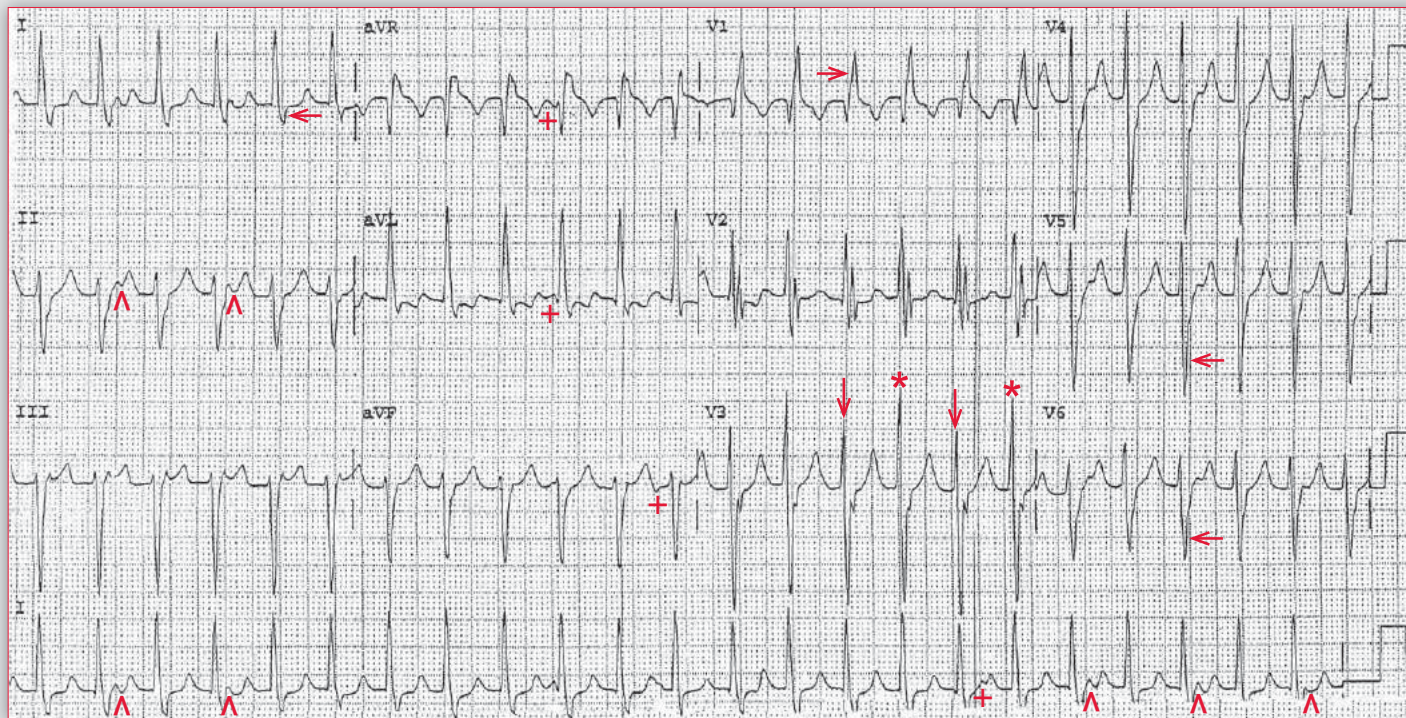
Practice Case 117

A 25-year-old medical resident develops acute onset palpitations after a busy call night and while on rounds with his attending. Other than the palpitations, the student has no other symptoms including chest pain, dyspnea, or pre-syncope. The attending measures his blood pressure to be 120/80, but his heart rate is at 140. The following ECG is obtained. The student has no baseline ECG for comparison.

What is the rhythm abnormality and its mechanism?

How would you initially manage this patient?





ECG 117 Analysis: Fascicular ventricular tachycardia, left ventricular tachycardia, electrical alternans

ECG 117 shows a regular, wide complex tachycardia at a rate of 140 bpm. The QRS complex duration is increased (0.12 sec) and it has a morphology of a right bundle branch block with broad R wave in lead V1 (→) and broad S waves in leads I and V5–V6 (←). The axis is extremely leftward between -30° and -90° with a positive QRS complex in leads II and aVF with an rS morphology. This is a left anterior fascicular block. The QT/QTc intervals are prolonged (320/490 msec) but are normal when the prolonged QRS complex duration is considered (300/450 msec).

There are no obvious P waves seen before each of the QRS complexes. However, there are positive waveforms seen in the lead I rhythm strip before the tenth, twelfth, and eighteenth QRS complexes (+) that look like P waves, although there is variability in the PR intervals, *ie*, they are dissociated. In addition there is random variability in the ST-T waves seen (^), which may represent superimposed P waves or changes in repolarization. In fact, independent P waves can be seen marching through the entire lead I rhythm strip. Therefore the presence of AV dissociation is consistent with a diagnosis of ventricular tachycardia.

The QRS complex is not very wide and exhibits a morphology of a right bundle branch block and a left anterior fascicular block. This

is a pattern that is seen with a left posterior fascicular tachycardia (also termed a left ventricular tachycardia). It is a form of ventricular tachycardia that is likely reentrant, involving the distal portion of the left posterior fascicle as well as the ventricular myocardium. Since the ventricular tachycardia involves the left posterior fascicle, the QRS complex is relatively narrow and the arrhythmia is often confused with a supraventricular tachycardia. However, there is AV dissociation, confirming that it is ventricular in origin.

An additional finding is QRS alternans, best seen in lead V3 (*,↓). Although this may be seen with rapid supraventricular tachycardias and not usually in ventricular tachycardia, this form of a ventricular tachycardia is more organized, as part of the circuit involves the left posterior fascicle.

A fascicular tachycardia has been shown to be responsive to verapamil. Hence it is often termed verapamil-sensitive ventricular tachycardia. It also has been called Belhassen ventricular tachycardia. It usually occurs in those with a structurally normal heart and hence does not carry a risk for sudden death. Treatment is often with verapamil or a β -blocker. However, since this form of ventricular tachycardia usually occurs in younger patients without structural heart disease, ablation is frequently the therapy of choice. ■

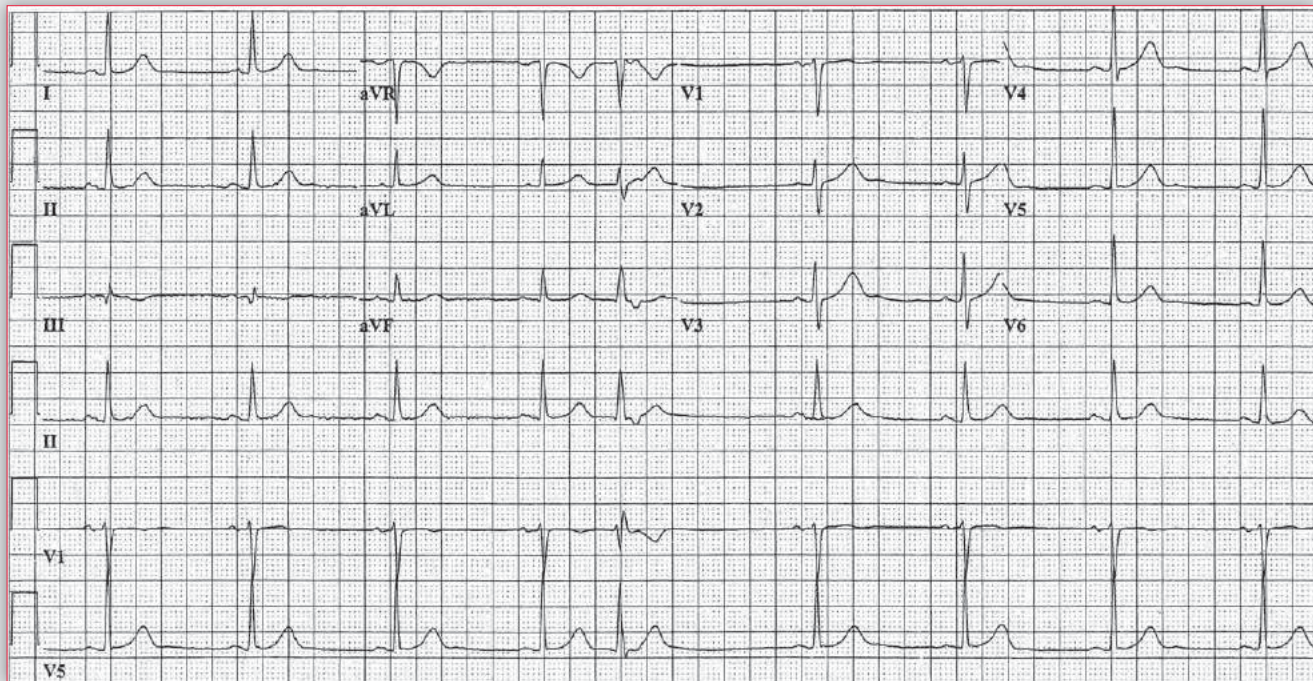
Notes

Practice Case 118

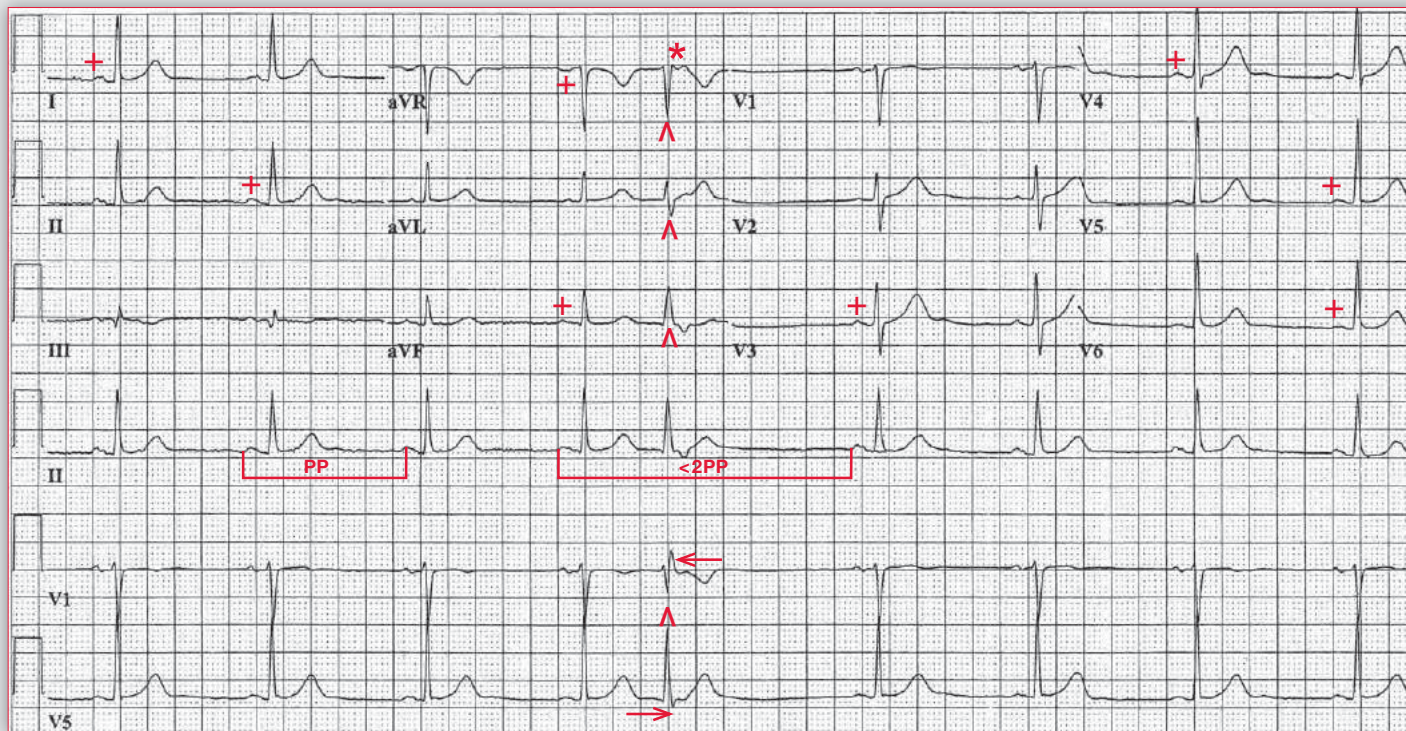
The following ECG is obtained during a routine pre-employment health visit for a pilot.

What is the abnormality?

Does this finding require any further cardiac evaluation?



Podrid's Real-World ECGs



ECG 118 Analysis: Sinus bradycardia, premature junctional complex with rate-related aberration

ECG 118 shows a regular rhythm at a rate of 54 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus bradycardia. The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis between 0° and +90° (positive QRS complex in lead I and aVF). The QT/QTc intervals are normal (400/380 msec).

The fifth QRS complex is premature (^), and it has a prolonged duration (0.14 sec). The morphology is typical for a right bundle branch block with an RSR' morphology in lead V1 (←) and a broad S wave in V5 (→). There is no P wave before this QRS complex. There is a P wave seen after the QRS complex, best seen in lead aVR (*). It is therefore a premature junctional complex with a rate-related right bundle branch block aberration. Although it might be considered a premature ventricular complex, the QRS complex has a typical right bundle branch block morphology. In addition, the pause associated with this premature complex is less than a full compensatory pause,

ie, the PP interval around the pause is less than two sinus (PP) intervals (⊔), which supports a supraventricular and not ventricular origin. Premature ventricular complexes are associated with a full compensatory pause, *ie*, the PP interval around the premature complex is equal to two sinus (PP) intervals. This is because premature junctional complexes are likely to conduct through the atrium to reach and reset the sinus node. Indeed, a retrograde P wave is seen in lead aVR (*). Premature ventricular complexes do not usually alter sinus node function; thus, there is one-time sinus node activity, but the sinus impulse is not able to penetrate the AV node (it is blocked). The next on-time sinus impulse does get through to result in a QRS complex, accounting for the compensatory pause.

Premature junctional complexes are typically benign, asymptomatic, and not associated with structural heart disease. Further cardiac evaluation of this pilot is needed only if symptoms of arrhythmia develop in the future. ■

Notes

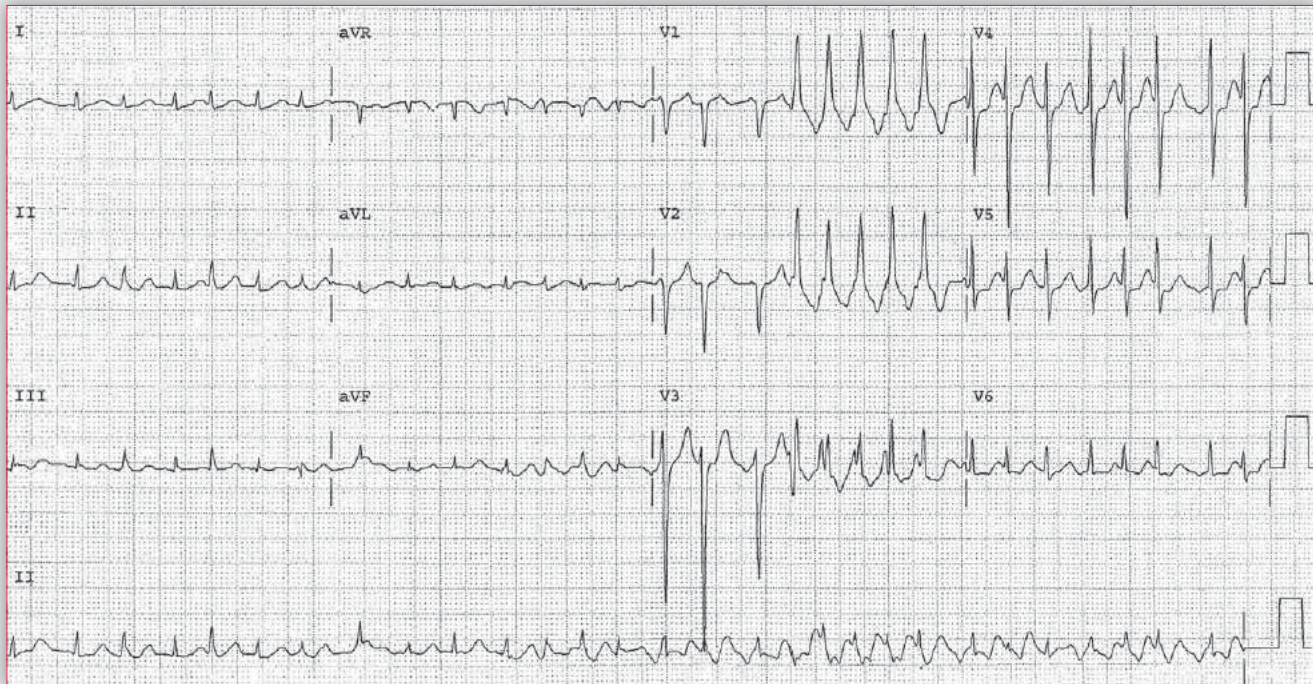
Practice Case 119

A 28-year-old woman from Africa with rheumatic mitral stenosis and chronic exercise limitation presents with palpitations and pre-syncope. While you are examining her, you note that the heart rate is highly variable and quite tachycardic. The following ECG is obtained.

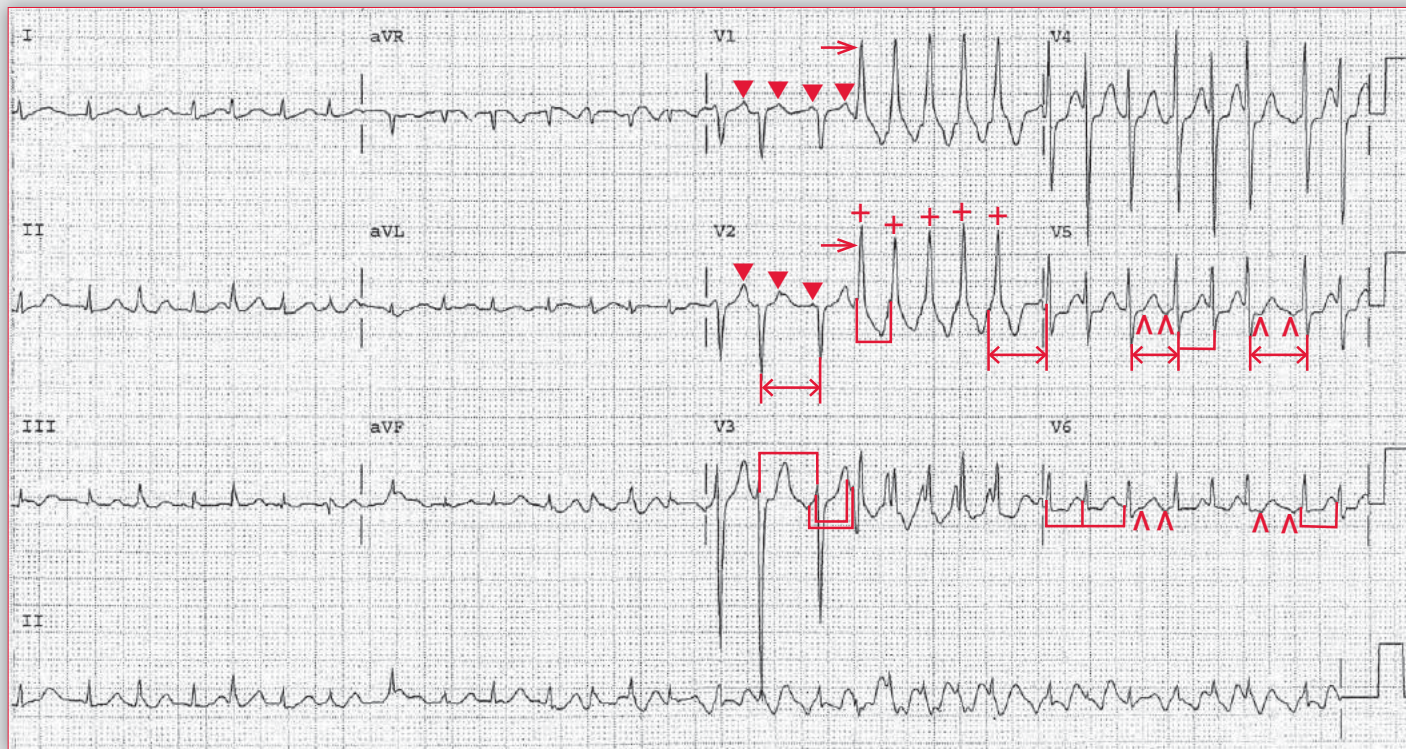
What is the rhythm abnormality?

What is causing the wide range in ventricular rates?

What is the mechanism of the wide QRS complexes?



Podrid's Real-World ECGs



ECG 119 Analysis: Atrial fibrillation, atrial flutter with 1:1 AV conduction (and intermittent 2:1 AV conduction), low voltage in the limb leads, Ashman phenomenon

ECG 119 shows the rhythm in the initial portion of the ECG (first 15 QRS complexes) is irregularly irregular at a rate of approximately 180 bpm. There are three arrhythmias that are irregularly irregular, including sinus arrhythmia, in which there is one P-wave morphology and PR interval; multifocal atrial rhythm (wandering atrial pacemaker) (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm), in which there are > 3 distinct P-wave morphologies without a dominant P-wave morphology and variable PR intervals; and atrial fibrillation, in which there is no organized atrial activity or P wave. In this ECG, there are no P waves seen. This is therefore atrial fibrillation. The second portion of the ECG shows a rhythm that is fairly regular (□) at a rate of approximately 260 bpm, although there are four longer RR intervals seen (↔). The three longer intervals are equal in duration (rate of 136 bpm). Although P waves are not obvious, there are positive waveforms seen in lead V1 and V2 (▼). They are occurring at a regular interval (rate 260 bpm) that is identical to the ventricular rate. The same atrial waveforms (^) are seen between the long RR interval at the end of the ECG in leads V5–V6; they have the same rate (260 bpm). Hence the rhythm during the second part of the ECG is atrial flutter and there is 1:1 AV conduction and four episodes of 2:1 AV conduction (↔).

Most of the QRS complexes are narrow (0.08 sec), and there is a normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). However, there is low voltage in the limb leads (< 5 mm in each lead). The QT/QTc intervals are normal (260/450 msec). The last 5 complexes (complexes 18–22) in leads V1–V3 (+) are wide (duration 0.12 sec), and they have a right bundle branch block morphology with a tall R wave in leads V1–V2 (→). The rate of these QRS complexes is 260 bpm; however, this is not a rate-related aberration as it can be seen that there are other QRS complexes that also have a rate of 260 bpm (□), yet they are not aberrated. It should be noted that these wide QRS complexes occur after a combination of long-short RR intervals (□, □). Hence these complexes manifest the Ashman phenomenon, which is a conduction block resulting from normal physiologic changes in refractoriness of the His-Purkinje system. When the heart rate is slow (long RR interval), membrane refractoriness is long, and when the heart rate is fast (short RR interval), refractoriness increases. When there is an abrupt change in rate (long-short RR interval), refractoriness does not adapt immediately, and hence one or several QRS complexes are conducted with aberration. Rate-related aberration or a functional bundle branch block, in which QRS complex widening is associated with a faster heart rate, results from an abnormality in conduction due to underlying disease of the His-Purkinje system. In this situation the aberration occurs when the heart rate is faster. ■

Practice Case 120

An 83-year-old man presented to an ophthalmology clinic for a preoperative evaluation before cataract surgery. He did not have any symptoms, but he was noted to have a rapid heart rate. Blood pressure was normal. However, he was sent to the emergency department. He denied any previous cardiac history. Physical examination was normal except for the rapid

ECG 120A



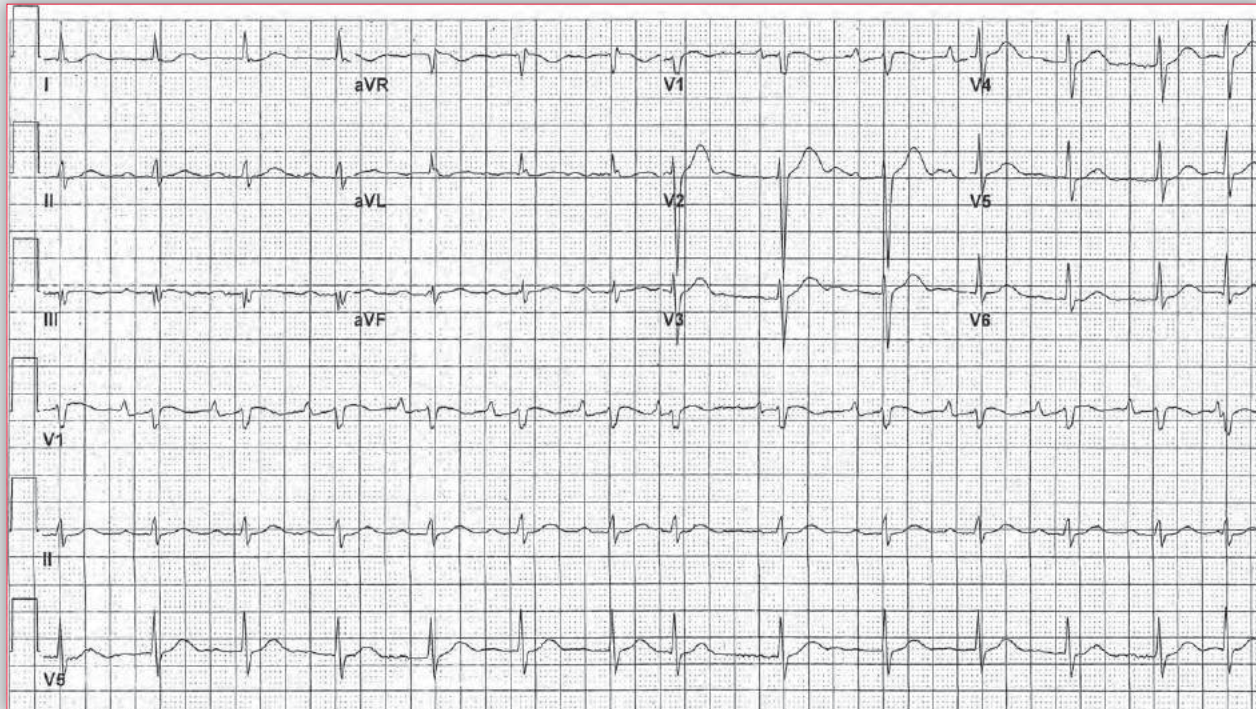
Practice Case 120

heart rate. An ECG was obtained (ECG 120A). It was felt that he had a supraventricular tachycardia and therapy with adenosine was given, without benefit. Thereafter, he was treated with intravenous β -blocker, verapamil, and then diltiazem. There was no change in the heart rate and ultimately he was cardioverted. An ECG was obtained after cardioversion (ECG 120B).

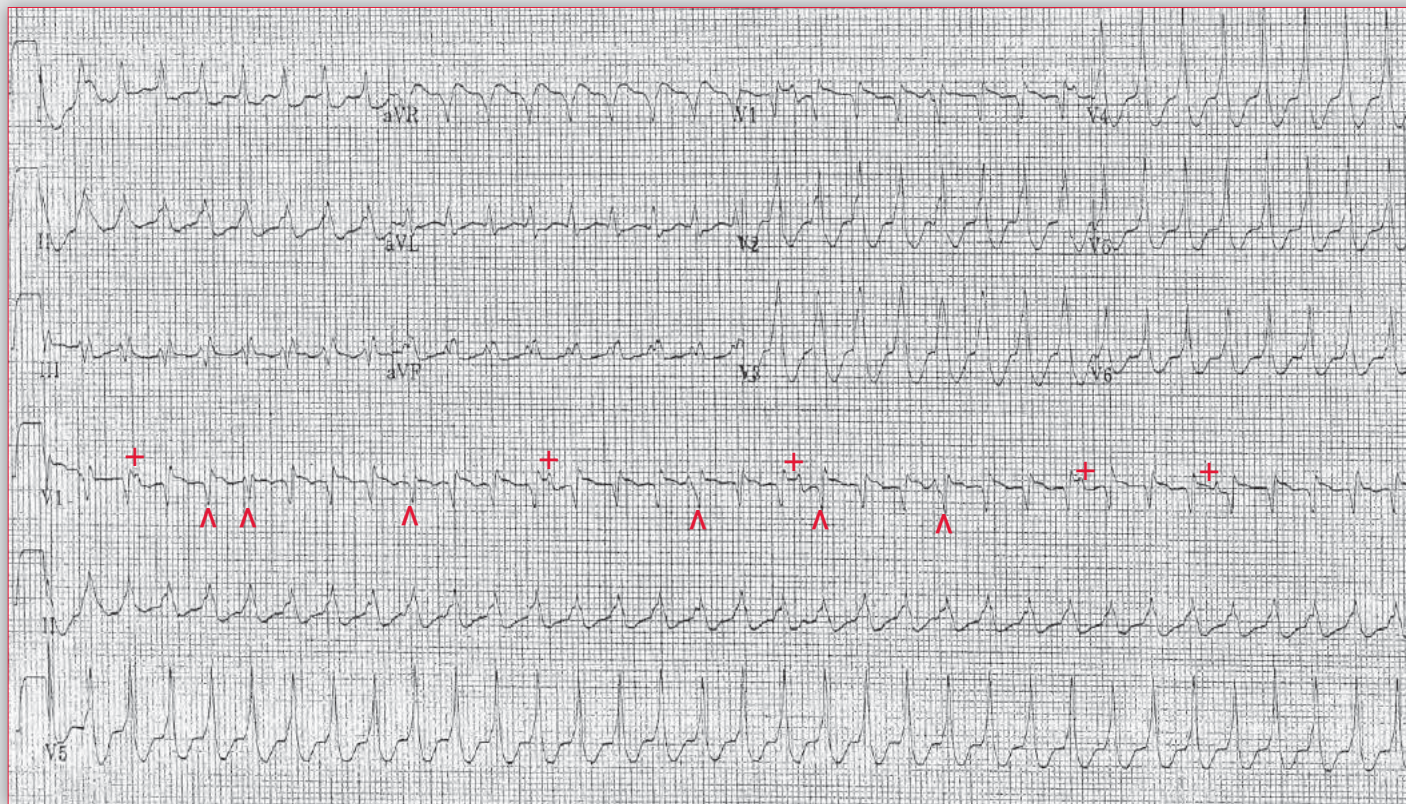
What is the etiology of the arrhythmia?

Was the treatment given appropriate?

ECG 120B



Podrid's Real-World ECGs



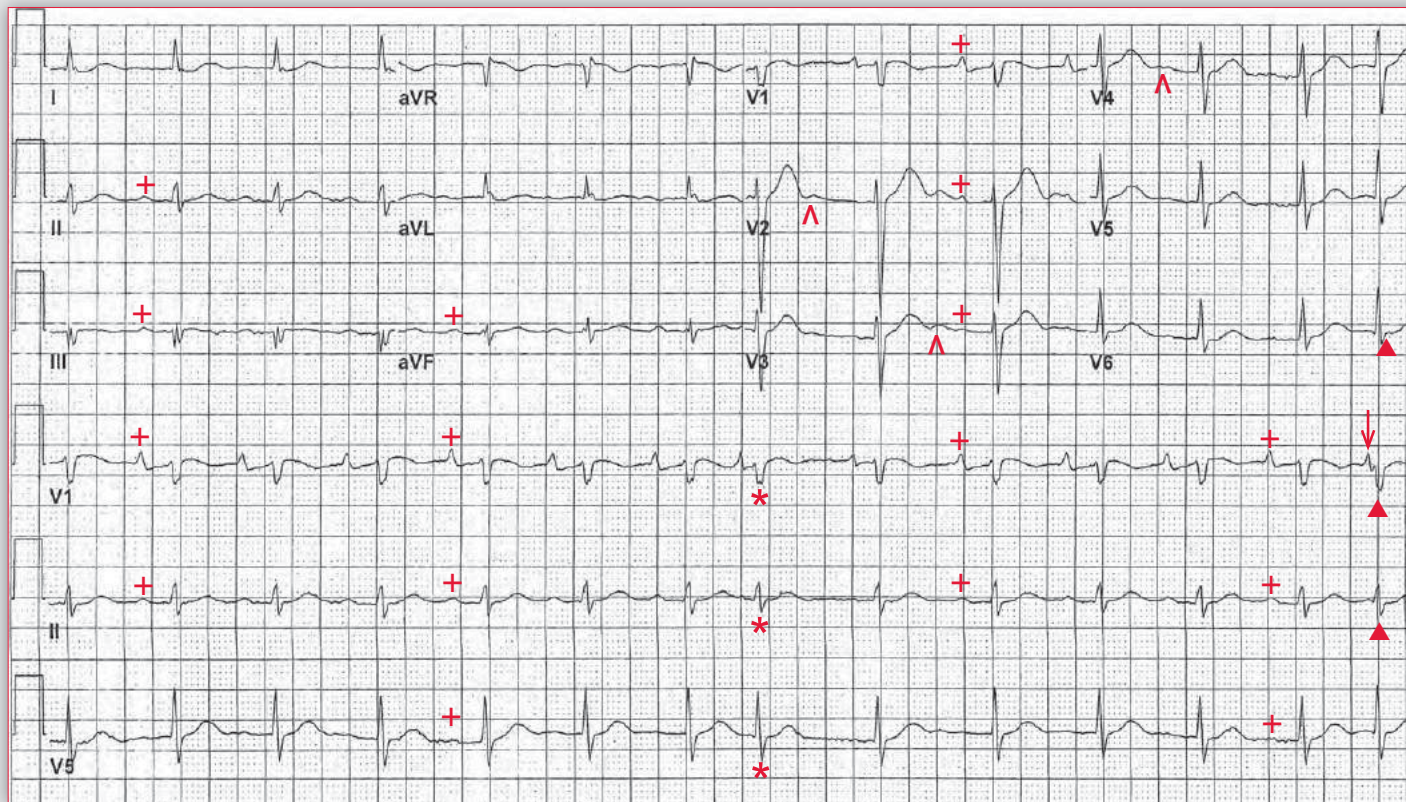
ECG 120A Analysis: Ventricular tachycardia

Although ECG 120A appears that this is a narrow QRS complex tachycardia (with narrow QRS complexes in several of the leads), it can be seen that this is actually a wide complex tachycardia at a rate of approximately 240 bpm. As seen in leads II, aVF, and V2–V3, the QRS complex duration is increased (0.14 sec), but the morphology is not typical for either a right or left bundle branch block. Although there are no obvious P waves seen, there are positive waveforms seen, especially

in lead V1 (after the second, twelfth, eighteenth, and twenty-fifth QRS complex and before the twenty-ninth complex) (+). These are P waves and they are not related to the QRS complex, *ie*, there is variability of the PR intervals that represent AV dissociation. In addition there is variability in the ST-T waves as well as variability in the QRS complex morphology, especially in lead V1 (^). These features are consistent with a diagnosis of ventricular tachycardia.

continues

Podrid's Real-World ECGs



ECG 120B Analysis: Normal sinus rhythm, first-degree AV block, right atrial hypertrophy, U waves, premature atrial complexes

ECG 120B is the baseline ECG for patient with ECG 120A. There is a regular rhythm at a rate of 80 bpm. There are P waves before each QRS complex (+) with a stable PR interval (0.26 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm with a first-degree AV block. The P wave is positive and very prominent in lead V1, suggesting a right atrial abnormality or hypertrophy. The QRS complexes have a normal duration (0.08 sec) and a normal morphology. The axis is normal at about 0° (positive QRS complex in lead I and biphasic in lead aVF). Prominent U waves (^) are seen in leads V2–V4.

The eighth QRS complexes is premature (*). The P wave has a slightly different morphology compared to the sinus P waves. The QRS complex has the same morphology as the sinus complexes. Hence this is a premature atrial complex. The last QRS complex is also premature (▲). Although there is a P wave before this QRS complex (↓), this is an on-time sinus P wave. The PR interval is very short and hence this P wave is nonconducted; rather this is a premature junctional complex.

When comparing the sinus complexes with those during the tachycardia, it can be seen that the complexes during the tachycardia are very different and not likely to be supraventricular with aberration. The etiology for a wide complex tachycardia *ie*, supraventricular tachycardia with aberration versus ventricular tachycardia is not based upon the clinical presentation, but rather based on close inspection of the

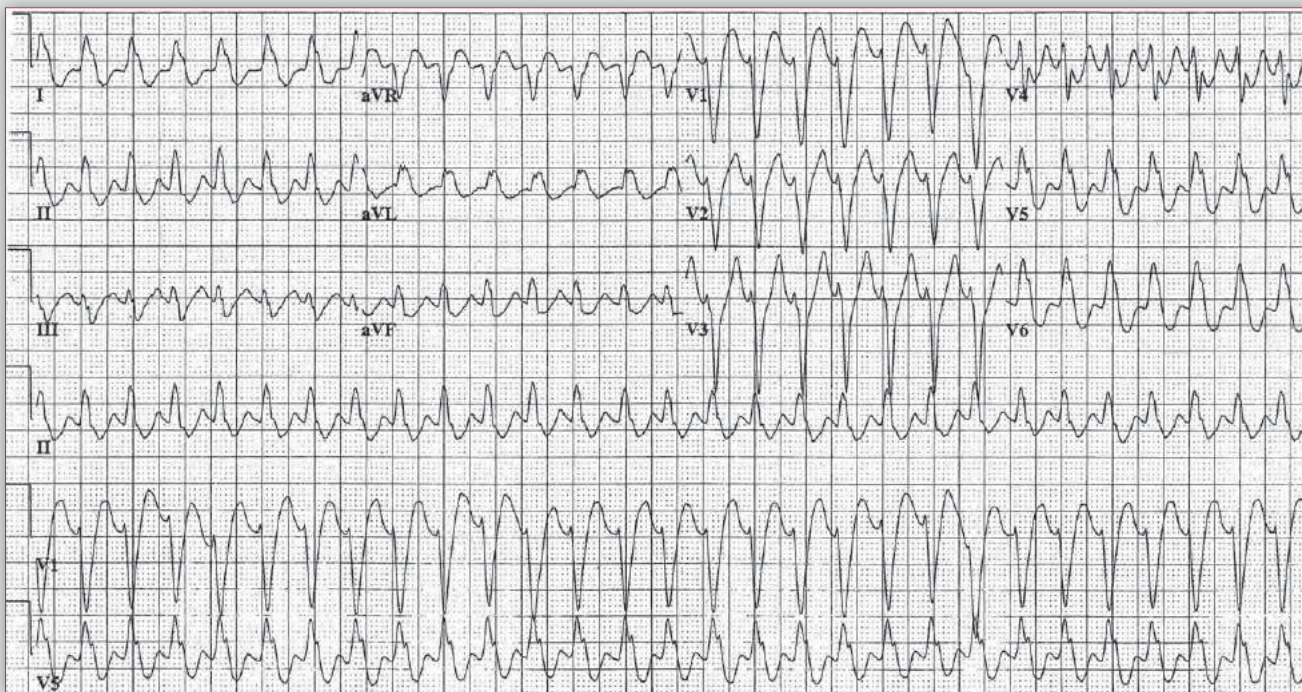
ECG. The clinical presentation is not based upon the nature of the arrhythmia, but rather on hemodynamic effects of the arrhythmia as it relates to the rate and the nature and extent of underlying heart disease and left ventricular function. It is not uncommon for a diagnosis of supraventricular tachycardia to be made whenever the patient is asymptomatic and has a normal blood pressure, as was seen in this case. It is of interest that even though this patient was 83 and had a ventricular tachycardia at a rate of 240 bpm, he remained asymptomatic. Also of interest is that he remained asymptomatic and there was no change in blood pressure despite therapy with adenosine, a β -blocker, verapamil, and diltiazem. These drugs, which are appropriate therapy for a supraventricular tachyarrhythmia as their major effect is on the AV node, should not be given for patients with ventricular tachycardia as they are not likely to work to revert the tachycardia. In addition, transient hypotension often occurring with these agents may result in acceleration of the rate of the ventricular tachycardia and may provoke ventricular fibrillation.

Appropriate acute therapy for ventricular tachycardia involves drugs that affect the ventricular myocardium, particularly class IA (especially IV procainamide), class IB agents (particularly IV lidocaine), or class III agents (especially IV amiodarone). Cardioversion is also an effective therapy, particularly if the patient is hemodynamically compromised. ■

Practice Case 121

An otherwise healthy 44-year-old man presents with acute onset of palpitations. He denies syncope or pre-syncope, and his blood pressure and oxygen saturations are stable. An ECG is obtained (ECG 121A). After therapy was given the arrhythmia terminated. A follow-up ECG was obtained (ECG 121B).

ECG 121A

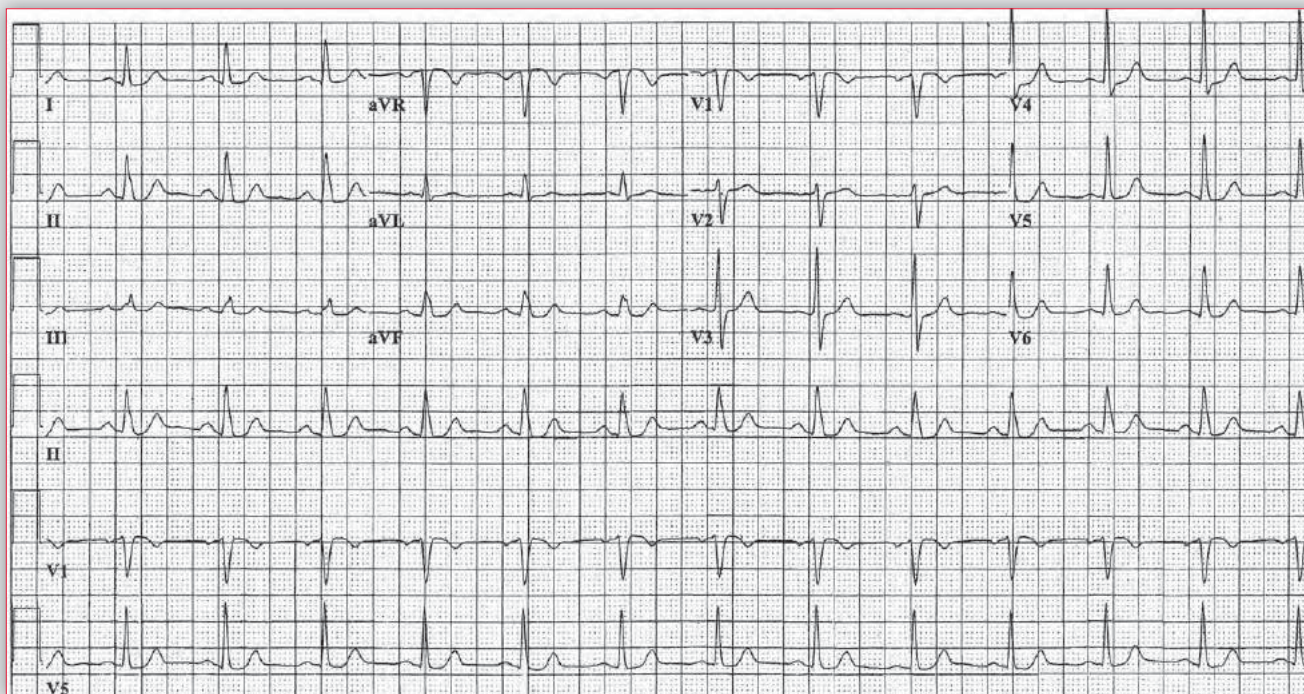


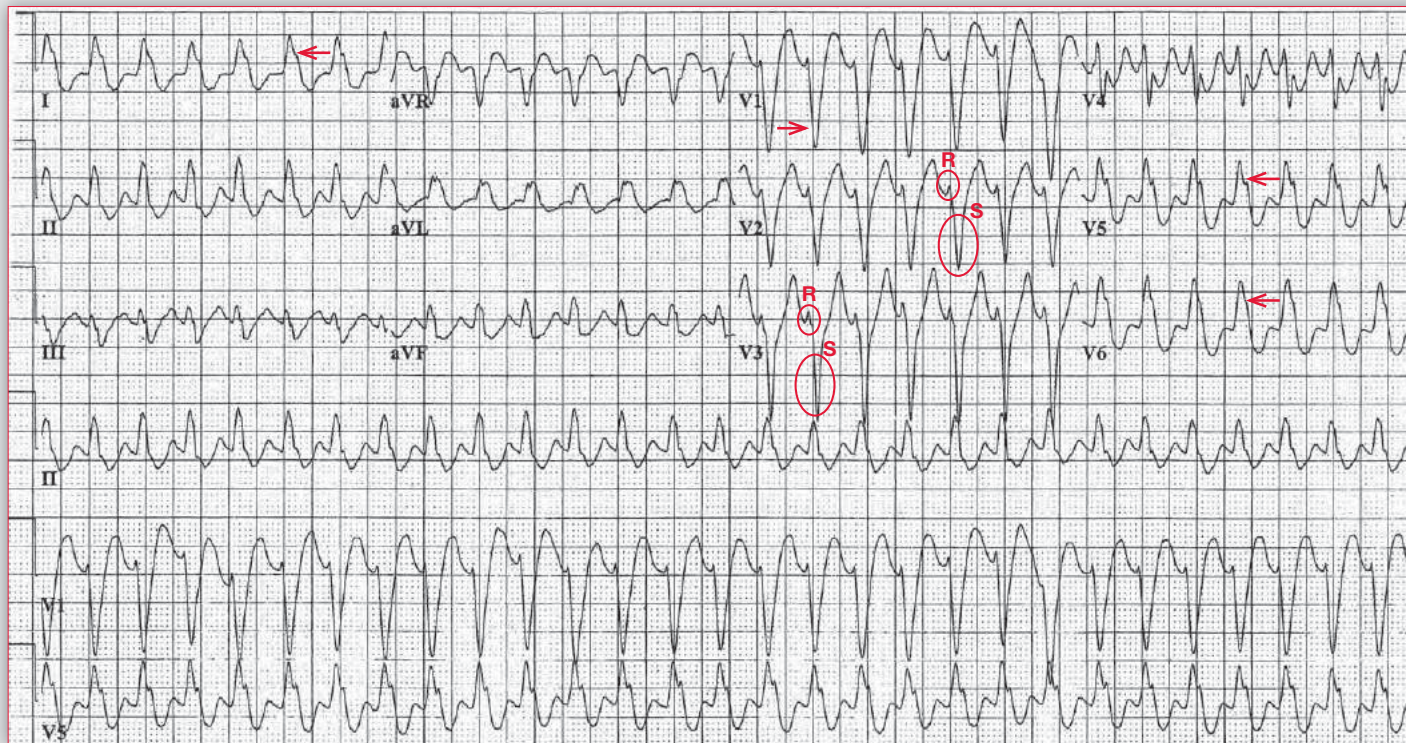
Practice Case 121

What is the etiology of the arrhythmia?

What would be appropriate therapy?

ECG 121B





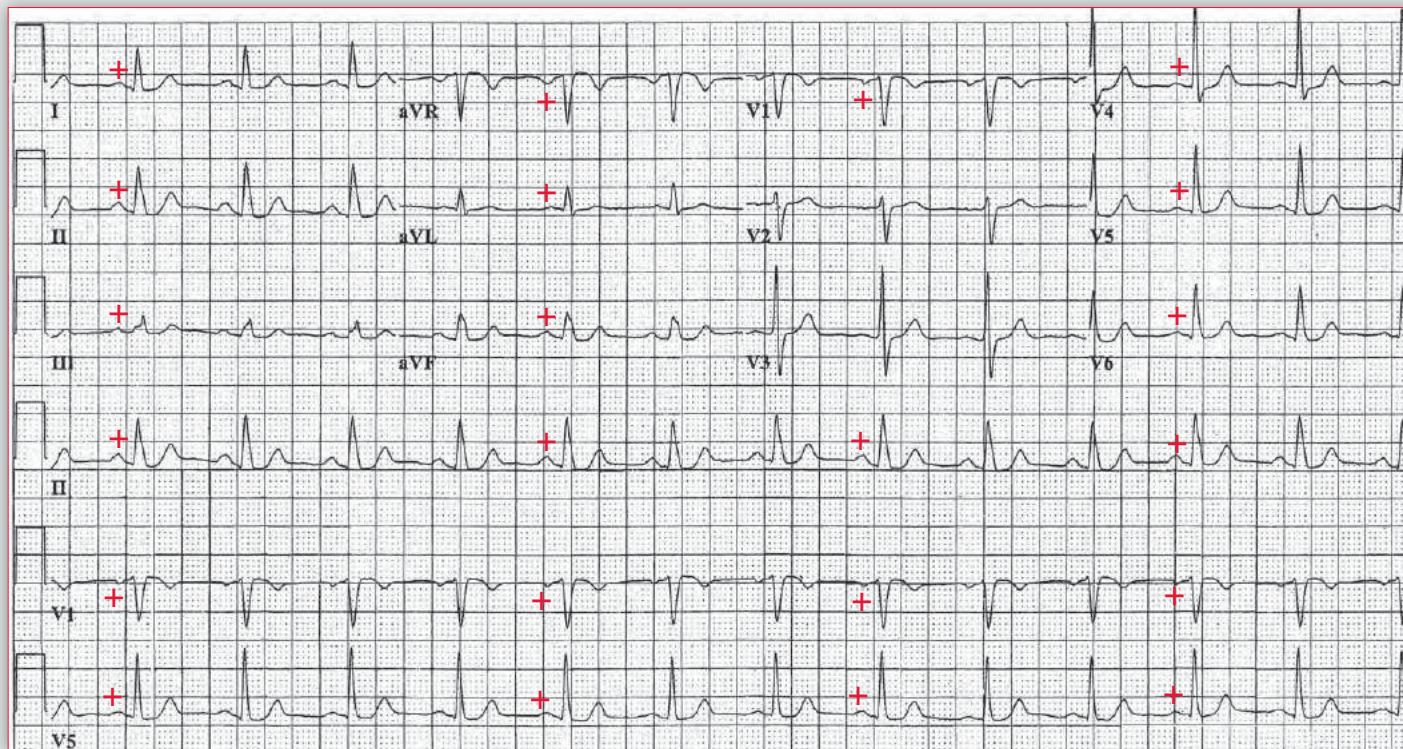
ECG 121A Analysis: Wide complex tachycardia, atrioventricular nodal reentrant tachycardia, rate-related left bundle branch block

ECG 121A shows a regular, wide complex tachycardia at a rate of 160 bpm. The QRS complex duration is increased (0.14 sec) and has a left bundle branch block morphology with a deep S wave in lead V1 (\rightarrow) and a broad R wave in leads I and V5–V6 (\leftarrow). The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (320/520 msec) but are normal when the prolonged QRS complex duration is considered (280/440 msec). There are no obvious P waves seen before or after any of the QRS complexes. The etiology of this arrhythmia is not immediately obvious, and it may be ventricular tachycardia or supraventricular tachycardia with a left bundle branch block aberration. The morphology is that of a typical left bundle branch block, favoring a diagnosis of supraventricular tachycardia. In addition, there is an R/S complex in leads V2–V3

and it can be seen that the R wave is < 100 msec and that the R/S ratio is < 1 ; these features favor a supraventricular origin of the tachycardia. Since aberration is due to a right or left bundle branch block, the initial activation of the ventricles is via the normal bundle and His-Purkinje system. Hence the R wave is narrow (< 100 msec) and narrower than the S wave, with ventricular tachycardia, all of ventricular activation is abnormal, bypassing the His-Purkinje system. In this situation, the R wave is also abnormal and wider than 100 msec and the R wave is often wider than the S wave. Therefore it is most likely that this is a supraventricular tachycardia with a left bundle branch block. As P waves are not present, this is a no-RP tachycardia, and the most frequent etiology for this is an atrioventricular nodal reentrant tachycardia (AVNRT).

continues

Podrid's Real-World ECGs



ECG 121B Analysis: Normal sinus rhythm

ECG 121B is the baseline ECG for the patient with ECG 121A. There is a regular rhythm at a rate of 78 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec) and there is a normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/410 msec). When comparing the QRS complex morphology seen in ECG 121A, it can be seen that the morphology and duration of the initial forces of the QRS complex are the same as in this ECG, with the prolonged QRS complex duration seen in ECG 121A being due to the terminal widening resulting from delayed terminal forces. This favors a diagnosis of supraventricular tachycardia for ECG 121A.

Therapy for an AVNRT involves alteration of the AV nodal electrophysiologic properties. This can be achieved with a vagal maneuver such as carotid sinus pressure or Valsalva. This is perhaps the safest form of therapy for a wide complex tachycardia of unknown etiology.

Even if the rhythm is ventricular, a vagal maneuver can be safely performed. In addition, by enhancing vagal tone, there may be a change in the sinus rate (PP interval), and this may result in exposing P waves that may not be otherwise seen.

Initial pharmacologic therapy for AVNRT is adenosine, which is often effective for terminating this arrhythmia. Although guidelines now state that this agent can be given for a wide complex tachycardia of unknown etiology (and hence even if the arrhythmia is ventricular tachycardia), this should be done with caution, as even transient hypotension caused by adenosine may accelerate the rate of a ventricular tachycardia or might provoke ventricular fibrillation. Verapamil or diltiazem (which may also cause hypotension) should not be administered when there is a wide complex tachycardia unless it is certain that the etiology is supraventricular. β -blockers are often given for patients with ventricular tachycardia, and although they are not likely to be effective, they are infrequently associated with an adverse reaction. ■

Notes

Index

A

antegrade-concealed conduction, 174–175, 242–243, 252–253
anteroapical myocardial infarction, 6–7
anterolateral myocardial infarction, 216–217
antidromic atrioventricular reentrant tachycardia, 42, 45
Ashman phenomenon, 32–33, 200, 202–203, 314–315
atrial arrhythmias, 39
atrial bigeminy, 112–113, 132–133
atrial fibrillation, 32–33, 70–71, 108–109, 136–137, 200–201, 302–303, 314–315
 Ashman phenomenon in, 203
 reversion of, to atrial flutter, 89
 from Wolff-Parkinson-White syndrome, 137, 139
atrial flutter, 232–233
 with 1:1 atrioventricular conduction, 48–50, 84–85, 196–197, 314
 paroxysmal, with 2:1 atrioventricular conduction, 256
 reversion of, to atrial fibrillation, 89
 with 3:1 atrioventricular conduction, 192–193
 with 3:12 Wenckebach, 194–195
 with 2:1 atrioventricular block, 36–37, 50–51, 148–149, 151, 166–167, 220, 242–243, 252–253, 286–287
 with 2:1 atrioventricular conduction, 28–29, 102, 192–193, 216, 256
 with variable atrioventricular conduction, 192–195, 258–259
 with variable block, 88–89
atrial tachycardia, 74–75, 248–249
 ectopic, 120–121, 142, 268–269
 multifocal, 116–117
 with variable atrioventricular block, 174–175
atrioventricular block
 atrial tachycardia with, 174–175
 first-degree, 6–7, 22, 24, 80, 154, 162–163, 212–213, 228–229, 264–265, 298–299, 320–321
 3:1, 242–243
 2:1, 36–37, 50–51, 120–121, 148–149, 151, 166–167, 220, 242–243, 252–253, 286–287

atrioventricular conduction
 1:1, 48–50, 84–85, 196–197, 314
 prolonged, 6–7, 22
 3:1, 192–193
 2:1, 28–29, 102, 192–193, 216, 256
atrioventricular dissociation, 45
atrioventricular nodal reentrant tachycardia (AVNRT), 10–11, 64–65, 92–93, 126–127, 154–157, 170, 178–179, 236, 272–273, 324–325, 327
 slow-slow, 17, 209, 237, 239
atrioventricular reentrant tachycardia
 antidromic, 42, 45
 orthodromic, 16–17, 282–283
atrioventricular sequential pacing, 232–233
AV. *See* specific atrioventricular entries

B

Belhassen ventricular tachycardia, 307
biatrial hypertrophy, 244–245
biventricular pacing, 302–304
bradycardia, sinus, 44–45, 80, 92–93, 278–280, 310–311
bundle branch blocks
 functional, 109, 112–113
 left. *See* left bundle branch block
 right. *See* right bundle branch block

C

clockwise rotation, 84–85, 88–89
concealed antegrade conduction.
 See antegrade-concealed conduction
counterclockwise rotation, 150–151, 178–179, 280–281

D

delta wave, 19, 279
diffuse nonspecific ST-T wave abnormalities, 122–123, 162
dual atrioventricular nodal pathways, 273

E

ectopic atrial rhythm, 122, 144–145
ectopic atrial tachycardia, 142, 268–269
 with 2:1 atrioventricular block, 120–121

ectopic junctional rhythm, 210–211, 296–297
ectopic junctional tachycardia, 65, 208, 211, 294–295, 297
electrical alternans, 16–17, 84–85, 170–171, 248–249, 306–307

F

fascicular block
 left anterior, 10–13, 32–33, 58–61, 64–67, 84–85, 116–117, 178–179, 192–197, 252–253, 307
 left posterior, 109, 142–144
fascicular ventricular tachycardia, 306–307
first-degree atrioventricular block, 6–7, 22, 24, 80, 154, 162–163, 212–213, 228–229, 264–265, 298–299, 320–321
functional bundle branch block, 109, 112–113

H

hyperkalemia, 160–161, 163

I

infarction. *See* myocardial infarction
inferior myocardial infarction, 102–105, 126–129, 220, 222–223
inferior wall myocardial infarction, 88–89, 263
interpolated premature atrial complexes, 132–133
intraventricular conduction delay, 80–81, 88–89, 192–195, 282–283
 to right ventricle, 208–213
 with use dependent effect, 196–197
IVCD. *See* intraventricular conduction delay

J

junctional escape complex, 256–257

L

LAAB. *See* left anterior fascicular block
late transition, 88–89
lateral myocardial infarction, 6–7
lateral wall myocardial infarction, 252–253

LBBB. *See* left bundle branch block
 left anterior fascicular block, 10–13, 32–33, 58–61, 64–67, 84–85, 116–117, 178–179, 192–197, 252–253, 307
 left atrial hypertrophy, 38, 98–99, 226, 228, 244–245, 278–280
 left axis, 126–129, 132–133, 178–179
 left bundle branch block, 22–24, 36, 38, 96–99
 rate-related, 48, 50, 108–109, 184–187, 226, 256–259, 324–325
 with ST-T wave abnormalities, 226–227
 left posterior fascicular block, 109, 142–144, 216
 left ventricular aneurysm, 6–7
 left ventricular hypertrophy, 70–71, 88–89, 178–180
 with ST-T wave abnormalities, 132–133
 left ventricular tachycardia, 306–307
 long PR tachycardia, 22, 36
 long RP tachycardia, 142–143, 148, 248–249, 282–283, 286–287
 low voltage, 10–11, 208–209
 in limb leads, 6–7, 32–33, 84–85, 216–217, 314–315
 in precordial leads, 126–129
 LPPB. *See* left posterior fascicular block

M

MI. *See* myocardial infarction
 multifocal atrial tachycardia, 116–117
 myocardial infarction
 anteroapical, 6–7, 216
 anterolateral, 216–217
 inferior wall, 88–89, 263
 lateral, 6–7
 lateral wall, 252–253
 old, 6–7, 88–89, 102–105, 126–129, 216–217, 220, 222–223, 252–253
 pseudo inferior wall, 279

N

narrow complex supraventricular tachycardia, 10–11, 64–65, 84–85, 92–93, 126–127, 154–155, 170–171, 272–273, 286
 nonspecific ST-T wave abnormalities, 80–81, 268–269
 diffuse, 122–123, 162

nonspecific T-wave abnormalities, 222–223
 no-RP tachycardia, 10–11, 64, 92–93, 126–127, 156, 170–171, 178–179, 272–273, 295, 325

O

orthodromic atrioventricular reentrant tachycardia, 16–17, 282–283

P

P-wave activated ventricular pacing, 232–233
 pacemaker-associated tachycardia, 232–233, 302–303
 pacing
 atrioventricular sequential, 232–233
 biventricular, 302–304
 P-wave activated ventricular, 232–233
 pacing associated tachycardia, 28–29
 paroxysmal atrial flutter with 2:1 atrioventricular conduction, 256
 posteroseptal accessory pathway, 278–279
 PR interval, 93, 97, 99, 175, 295
 precordial leads, low voltage in, 126–129
 premature atrial complexes, 38–39, 58–59, 67, 92–93, 272–273, 280–281, 320–321
 in bigeminal pattern, 112–113, 132–133
 interpolated, 132–133
 with preexcitation, 280–281
 with rate-related left bundle branch morphology, 184–185
 premature junctional complex with rate-related aberration, 310–311
 premature ventricular complexes, 88–89, 170–171, 200–201, 208–209, 294–295, 302–303
 prolonged atrioventricular conduction, 6–7, 22
 pseudofusion, 232–233, 302–303
 pseudoinfarction, 279
 PVC. *See* premature ventricular complexes

Q

QRS alternans, 170–171, 248–249, 307
 QT prolongation, 222–223

R

rate-related left bundle branch block, 48, 50, 108–109, 184–187, 226, 256–259, 324–325
 rate-related right bundle branch block, 112–113, 126–127
 RBBB. *See* right bundle branch block

reentry, 79
 right atrial hypertrophy, 6–7, 244–245, 320–321
 right axis, 42–43, 143
 right bundle branch block, 58–61, 66–67, 102–105, 220
 Ashman phenomenon as cause of, 32–33
 rate-related, 112–113, 126–127
 right bundle branch-like morphology, 42, 64–65
 right ventricle
 hypertrophy of, 116–117
 intraventricular conduction delay to, 208–213
 right ventricular conduction delay, 281

S

Sgarbossa criteria, 263
 short RP tachycardia, 16–17, 19, 208, 217, 236, 294–295
 sinoventricular rhythm, 161
 sinus arrhythmias, 298–299
 sinus bradycardia, 44–45, 80, 92–93, 278–280, 310–311
 sinus rhythm
 with first-degree atrioventricular block, 154, 264–265, 321
 irregular, 117, 137, 299
 normal, 6–7, 12–13, 18–19, 24–25, 38–39, 60–61, 66–67, 74–75, 98–99, 104–105, 112–113, 128–129, 132–133, 138, 150–151, 160–163, 180–181, 184–185, 212–213, 222–223, 228, 244–245, 272–273, 288–289, 320–321, 326–327
 sinus tachycardia, 22–23, 25, 58–59, 96–97, 99, 226–227, 238–239
 with rate-related left bundle branch block, 186–187
 with Wolff-Parkinson-White pattern, 54–55
 slow-fast atrioventricular nodal reentrant tachycardia, 11, 295
 slow-slow atrioventricular nodal reentrant tachycardia, 17, 209, 237, 239
 ST-segment depression, 85
 ST-T wave abnormalities
 left bundle branch block with, 226–227
 left ventricular hypertrophy with, 132–133
 nonspecific, 80–81, 122–123, 162, 268–269
 supraventricular tachycardia, 23, 148–149, 178–179, 208–209, 236, 294–295
 long RP, 142–143
 narrow complex, 10–11, 64–65, 84–85, 92–93, 126–127, 154–155, 170–171, 272–273, 286
 sustained monomorphic ventricular tachycardia, 4–5, 7, 79

T

tachycardia

- atrial. *See* atrial tachycardia
- atrioventricular nodal reentrant, 10–11, 17, 64–65, 92–93, 126–127, 154–157, 170, 178–179, 209, 236–237, 239, 272–273, 324–325, 327
- atrioventricular reentrant. *See* atrioventricular reentrant tachycardia
- ectopic junctional, 65, 208, 211, 294–295, 297
- long PR, 22, 36
- long RP, 142–143, 148, 248–249, 282–283, 286–287
- no-RP, 10–11, 64, 92–93, 126–127, 156, 170–171, 178–179, 272–273, 295, 325
- pacemaker-associated, 232–233, 302–303
- pacing associated, 28–29
- short RP, 16–17, 19, 208, 217, 236, 294–295

- sinus, 22–23, 25, 54–55, 58–59, 96–97, 99, 226–227
- supraventricular. *See* supraventricular tachycardia
- ventricular. *See* ventricular tachycardia
- verapamil-sensitive ventricular, 307
- wide complex, 22, 36–37, 42, 48, 78–79, 96–97, 321, 324–325

- T-wave alternans, 170–171, 248–249
- T-wave inversions, 113, 223

U

- U waves, 280–281, 320–321
- in V2–V3, 212–213

V

- ventricular flutter, 49
- ventricular pacing, P-wave activated, 232–233
- ventricular parasystole, 200–201

- ventricular tachycardia, 78–79, 262–263, 265, 318–319
 - fascicular, 306–307
 - left, 306–307
 - sustained monomorphic, 4–5, 7, 79
- verapamil-sensitive ventricular tachycardia, 307

W

- wide complex tachycardia, 22, 36–37, 42, 48, 78–79, 96–97, 321, 324–325
- Wolff-Parkinson-White (WPW) pattern, 44–45, 136–139, 278–279
 - sinus tachycardia with, 54–55
- Wolff-Parkinson-White (WPW) syndrome, 18–19
 - atrial fibrillation associated with, 137, 139

Notes